FUNGOUS DISEASES OF PLANTS

JAKOB ERIKSSON

SECOND EDITION

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FUNGOUS DISEASES OF PLANTS

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FUNGOUS DISEASES OF PLANTS

IN AGRICULTURE, HORTICULTURE AND FORESTRY

BY

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SECOND EDITION

TRANSLATED FROM THE GERMAN BY

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PREFACE TO THE SECOND ENGLISH EDITION

THIS volume presents a general review of the more important fungous diseases which occur in Northern and Mid-Europe on field and garden crops and on certain trees of economic or ornamental value. Alongside the description of the various diseases are given the methods to be adopted for their prevention and control. Some of the diseases described are, so far, only known in other countries, but in view of the ease of communication at the present time and the interchange of commodities between nations it is to be expected that these diseases may at any time make their appearance in Northern and Mid-Europe.

Some new material which was not available at the time of the publication of the German edition (1927–28) is included in this edition, more particularly in the chapter on cereal- and grass-rusts and in connection with certain diseases on peach, chestnut, and elm trees.

The views of different investigators are often at variance as to the nature and development of the organisms which produce diseases and also in regard to the methods of control. In such cases the reasons for these differences are stated briefly and objectively so that the reader may, in the light of his own experience, be in a position to form an independent opinion upon the points at issue.

To assist the reader who may wish to extend his study of phytopathology, references to some of the chief publications follow the descriptions of the chief groups of fungi and forms of disease. With the same object in view the names of investigators and the year of publication of their work are given in parentheses in the text. It thus becomes easy by referring to such publications as *Phytopathology*, *Journal of Agricultural Science*, *Review of Applied Mycology*, *Zeitschrift für Pflanzenkrankheiten*, *Centralblatt für Bakteriologie*, Abt. II., *Annales des Epiphyties*, *Revue de Pathologie Végétale*, etc., to find the original publication or an abstract of it.

In dealing with the description of certain diseases of trees in southern countries, concerning which my own knowledge was inadequate, I have been privileged to draw upon the very valuable account given by my friend Professor Etienne Foëx, Paris, of the forms of disease occurring on olive and chestnut trees in France and neighbouring countries. I take this opportunity of expressing to Professor Foëx my warmest thanks for the valuable assistance he has afforded to me.

The illustrations in this volume have been prepared either from my own drawings or photographs or taken from other publications with the consent of the publisher or author; in the latter case acknowledgment is given in the descriptive text.

I am much indebted to my friend Dr. Axel Lagrelius, of Stockholm, for the help he has afforded in connection with the illustrations, and I express to him my very sincere thanks.

I wish also to thank the translator, Dr. William Goodwin, for the pains he has taken in the preparation of this translation, and especially for the ability with which he has interpreted and presented my personal views on numerous critical questions.

I am conscious of the numerous deficiencies of my book; some of them are attributable to the almost insuperable difficulty of obtaining access to original publications. I hope, however, that in spite of its deficiencies the book will assist in the waging of an active and successful campaign against plant diseases.

JAKOB ERIKSSON.

STOCKHOLM,
December, 1929.

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FUNGOUS DISEASES OF PLANTS

INTRODUCTION

THE STRUCTURE AND NATURE OF FUNGI

The fungi form a large, self-contained group of the plant kingdom. Outwardly they differ from the higher plants, as do the algæ and lichens, in not being divided into root, stem, and leaf. The body of the fungus consists of a thallus which varies in shape and structure. The internal construction and mode of life of the fungi differ from those of all other plant groups owing to the fact that the fungi are not provided with chlorophyll, and they are, consequently, unable to assimilate inorganic substances such as carbon dioxide, water, nitric acid, and ammonia, and convert them into organic compounds. They are thus obliged to obtain their food in a prepared form from other living or dead organisms. Fungi which derive their nourishment from living plants or animals are known as parasitic fungi, while those that live upon the organic remains of animals or plants are termed saprophytic fungi. The division between these two groups is not, however, a sharp one, as there are fungi which at one time live as parasites and at another as saprophytes.

That part of the body of the fungus which takes up and distributes the food is termed the vegetative system. As a rule, this is a mycelium consisting generally of septate and branched filaments or hyphæ (*Eumycetes*). In the lowest forms of these fungi, the mycelium is reduced to a single, usually non-septate, hypha (*Chytridiaceæ*). The *Schizomycetes* (bacteria) have no mycelium, and in the *Myxomycetes* (slime fungi) the mycelium is replaced by an irregularly-shaped, slime-like body, the plasmodium.

It is the duty of the fructificative system to provide for the spread of the fungus during the vegetative period, and to enable it to live from year to year. Dissemination takes place by means of thin-walled summer spores (uredospores, pycnoconidia) which germinate rapidly. The fungus is carried on from one year to the next by means of thick-walled winter spores (oospores, teleutospores, ascospores) which only germinate after over-wintering out of doors. Over-wintering is also provided for by means of dense, hard agglomerations of mycelium (sclerotia), and by a latent stage in which the protoplasm of the fungus is united symbiotically with that of the host cell, (mycoplasm).

The fungi have been classified according to a number of systems. In this volume, the classification given below has been adopted. The various fungous diseases which are described are arranged in accordance with this classification :—

- SCHIZOMYCETES—Bacteria
- MYXOMYCETES—Slime Fungi
- EUMYCETES—True Fungi
  - Oomycetes
    - Chytridiaceæ
    - Peronosporaceæ
  - Basidiomycetes
    - Ustilaginaceæ
    - Uredinaceæ
    - Hymenomycetineæ
    - Exobasidiineæ
  - Ascomycetes
    - Exoascaceæ
    - Erysiphaceæ
    - Perisporiaceæ
    - Hypocreales
    - Dothideales
    - Sphæriales
    - Discomycetes
- FUNGI IMPERFECTI—Imperfect Fungi
  - Sphæropsidaceæ
  - Melanconiaceæ
  - Hyphomycetes
  - Fusarium
  - Rhizoctonia
  - DISEASES NOT COMPLETELY INVESTIGATED

CHAPTER I

SCHIZOMYCETES—BACTERIA

BACTERIA are the smallest and simplest of all living organisms. When aggregated in millions they may be observed as turbid masses in putrefying liquids and in diseased animal and vegetable tissues, but individually they can only be seen with the aid of a powerful microscope. Each bacterium consists of a single cell; frequently, however, several individuals are joined together in a colony and it is the aggregation of these colonies that produces the turbid appearance referred to above. Bacteria may be spherical, rod-shaped, and curved or spiral, and it is mainly the rod-shaped forms which are concerned in plant diseases.

As a rule, bacteria multiply by direct division (fission) into two portions, each of which can at once separate and begin an independent existence. Sometimes the individuals remain connected and give rise to colonies of various forms—thread-like, tabular, cubical. Bacteria are often provided with flagella, the number of which varies. In many bacteria there is, in addition to fission, a method of reproduction by spores. These spores (endospores) arise within the cell and have thick walls; they may be termed resting-spores, and they possess considerable resistance to external influences.

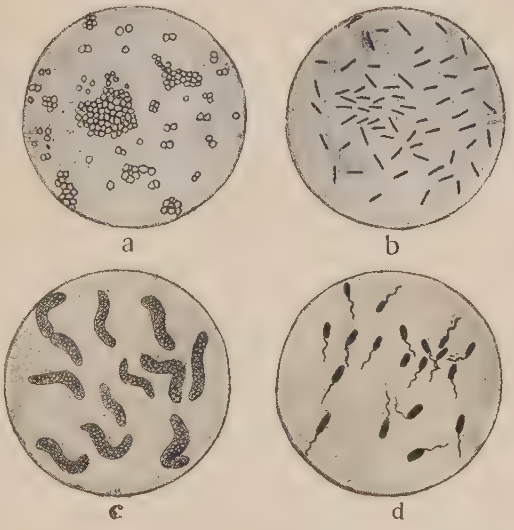

FIG. 1.—Forms of Bacteria.
a, spheres; *b*, rods; *c*, spirals; *d*, flagellate. Highly magnified. (After W. Migula and E. Warming.)

Latterly, several other phases of development have been described in the case of the bacteria, and amongst these is one termed "Bacterium-

Plasmodium," "Symplasm" in which the contents of one or more bacterial cells, together with the surrounding membranes, produces a symbiotic entity from which normal bacterial cells are subsequently generated. Finally, sexual processes have been reported as occurring amongst the bacteria (Almquist, 1923).

It has long been known that bacteria play a very important part in putrefaction and fermentation, as well as in the infectious diseases of human beings and animals. On the other hand, it is only during comparatively recent times that their connection with plant diseases has been recognised, and each year brings new discoveries in this field.

The bacteria which cause plant diseases belong mainly to the family of Bacteriaceæ (rod-shaped bacteria), which can be divided into three genera: (1) *Bacterium* Ehr., which are non-motile; (2) *Bacillus* Cohn., motile, with flagella distributed over the surface of the cell; (3) *Pseudomonas* Mig., motile, with one or more flagella at one end of the cell.

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Bacterial Rot of Potato

Bacillus solaniperda Mig. et al.

This disease appears in autumn at the time when the potatoes are being lifted. In the first stage, brown spots appear inside the tuber, either scattered irregularly throughout the tissue or in a large patch in the centre. In wet soil, especially clay, or if the potatoes have been stored when wet, the disease spreads through the tuber. Not only the cell walls but also the starch grains are acted upon until finally the potato is changed into an evil-smelling, slimy, grey mass surrounded by the skin (wet rot).

If, on the other hand, the weather is dry and the soil porous so that the water drains away, the decomposition ceases and the tuber drys up and becomes hard. Numerous small holes, surrounded by a layer of starch grains, arise inside the tuber (dry rot). Potatoes suffering from this form of the disease may be used for the manufacture of dextrin, etc.

For a long time the view was held that both wet rot and dry rot are caused by the potato blight fungus, *Phytophthora infestans* (Mont.), de By, and that the brown patches produced by the fungus on the surface of the ripening tubers are a preliminary stage of both forms of rot. It has, however, been finally decided that both wet and dry rot are caused by bacteria.

It is not yet clear which are the bacteria that cause these diseases, as the various investigators have arrived at different conclusions. On the one hand, the chief part in the destructive changes is ascribed to *Bacillus solaniperda* Mig., which belongs to the group of rod-shaped bacteria (Kramer, 1891). On the other hand, such bacterial forms as *Bacillus amylobacter* van Tiegh., *Bacterium fluorescens* Lehm. & Neum., *Bacterium xanthochlorum* Schust., *Bacterium phytophthorus* App. et al., are considered to act with *Bacillus solaniperda* in producing decomposition.

Infection takes place through a wound, and the bacteria live in the cells. The disease spreads from cell to cell, the vessels not playing an important part in its transference. On this account, the process of decomposition is localised, but usually ends in causing the early death of the organ that is attacked.

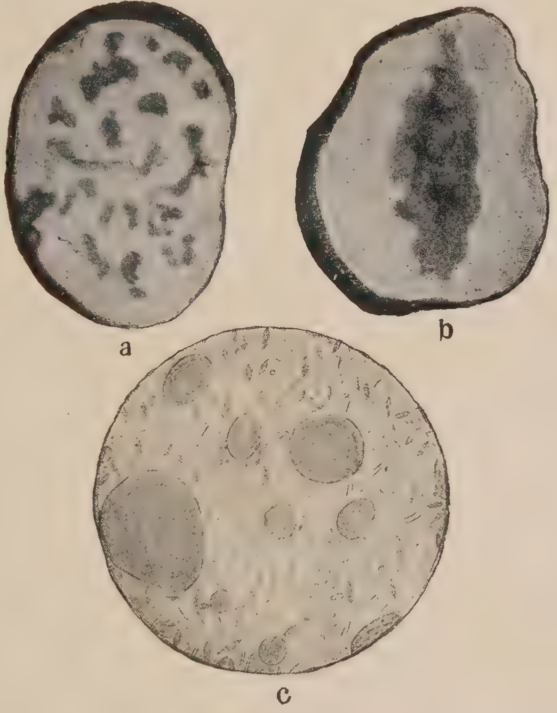

Fig. 2.—Bacterial Rot of Potato.

a and *b*, sections of diseased tubers ; *c*, portion of slime containing two forms of bacteria, *Bacillus amylobacter* with pointed ends, and *B. solaniperda* with rounded ends. (*a* and *b* after E. Rostrup, *c* after K. Wehmer.)

Some investigators are of the opinion that the healthy tissues of the tuber, whether whole or damaged, cannot be attacked by any form of bacterium in the first place, and it is only when such predisposing causes as excessive moisture or high temperatures (more than 15–20° C.) occur in the storage place that the normally harmless bacteria deliver their attack (Wehmer, 1898).

Some varieties of potatoes show a marked difference in their resistance to this disease. Early eating potatoes are, in general, the most susceptible, whereas the late sorts, which are used more for cattle feeding and industrial purposes, are the most resistant.

Control Measures :

(1) Healthy seed only should be used. (2) For cultivation on a large scale, those varieties should be chosen which have shown themselves locally to be resistant. (3) The potatoes should be grown on high, well-drained land ; if possible, a sandy soil should be chosen. (4) Adequate supplies of potash and phosphoric acid in the soil increase the resistance of the crop to disease ; heavy dressings of lime have the opposite effect. (5) The potatoes should be well dried before being stored, and the storage place should be dry and ventilated.

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Bacterial Wilt of Potato

Bacillus Solanacearum Erw. Sm. and *Bacterium sepedonicum* Spieck. & Kotth.

In 1906 a description was given of a new potato disease, "Bakterien-Ringkrankheit," occurring in West Germany, the following being the main characteristics : Some weeks after planting it was noticed that some of the seed had not produced plants. Examination showed in such cases that the seed tuber was apparently healthy but the shoots, as well as the root system, were dying. Some of the plants had grown above ground but had a sickly, dwarfed appearance with pale transparent stems and small leaves ; these plants finally ceased to grow. Others appeared healthy and grew normally until the middle of summer, when the haulms became transparent and spotted and withered prematurely.

This disease is caused by bacteria, especially *Bacillus Solanacearum*

Erw. Sm., which follow the fibro-vascular system throughout the plant and produce black spots or patches in the fibro-vascular ring; when the attack is slight a section of the potato shows the changes in the ring, but a severe attack causes the whole of the inside of the tuber to become black. Often the disease does not affect the external appearance of the potatoes.

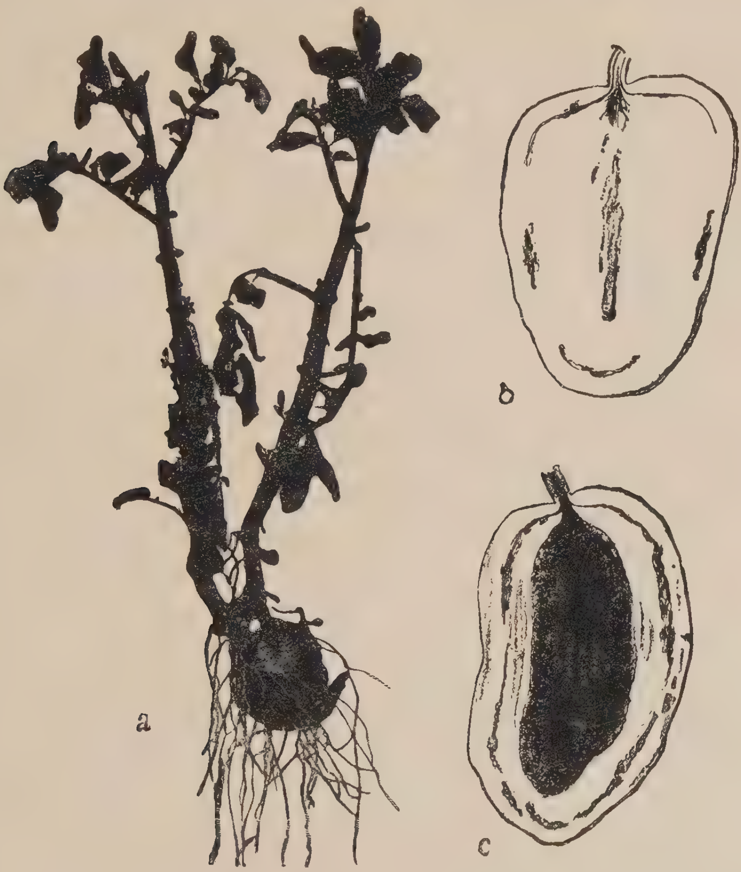

Fig. 3.—Bacterial Wilt of Potato.
a, plant grown from diseased tuber; *b*, slightly, *c*, severely diseased tubers in section. (After O. Appel.)

This disease is carried on from year to year through slightly infected tubers which, when used for seed, produce plants which are more or less diseased. The disease can be transferred to healthy potatoes if the bacteria can find entrance through a wound or injury.

The disease has sometimes had a devastating effect upon the potato crop. In 1905, for example, 60–70 per cent. was destroyed in one district, whilst in some cases the crop was not worth lifting. Many other plants such as tomato, tobacco, etc., are attacked by this same fungus (Stanford & Wolf, 1917).

In 1914 a new bacterial disease of the potato was described under the name of "Bakterien-Ringfäule." The fungus *Bacterium sepedonicum* Spieck. & Kotth. is responsible for this disease, which causes injury to the tuber and haulm. Outwardly the disease is not apparent on the tuber, but a section across the rose end of the potato shows softened places in the fibro-vascular ring ; sometimes the whole ring may pass into a state of wet rot. Through the destruction of the parenchyma surrounding the vascular bundles, there arise cavities which are filled with slimy material. When potatoes diseased in this way begin to grow the bacteria pass through the eyes into the young shoots. If the infection is severe the shoots die, or if they reach the surface they are stunted, both symptoms sometimes appearing on the same plant. When, however, infection is slight the foliage can apparently develop normally. Warm and dry weather seems to favour the development of the bacteria, and as a consequence the plants die more rapidly. The bacteria also invade the stolons and so lead to the infection of the new tubers and the carrying-on of the disease to the next year. In very dry years the disease is clearly evident in July, but in normal years it is not seen until the late summer. Potatoes suffering from this disease must be rejected for seed purposes.

Control Measures :

(1) Only healthy seed should be used, and it can be tested by dividing several tubers and exposing the cut surfaces to the air. If dark patches appear at once, or after an hour or two, the seed should not be used. (2) Whole sets are best for seed ; if cut sets are used they should be prepared a day or two beforehand, so that a corky layer may form on the cut surface and thus prevent the entrance of bacteria. (3) Potato fields should be inspected in summer and any suspicious, prematurely-withered plants should be removed—foliage as well as tubers.

Black-Leg of Potato

Bacillus phytophthorus App.

This disease makes itself apparent in early summer by a yellowing of the leaves of the potato plant and a cessation of growth, the upper leaves being often slightly incurved. At the same time the stem is seen to be black and beginning to die, the blackened portion not uncommonly being thinner than the healthy part; sometimes only a single stem of a plant is affected in this manner. Generally the diseased plants occur singly in a field, rarely in groups. The diseased stems stand loosely in the soil and can easily be drawn out. In wet weather the blackened portion of the stem is slimy to the touch.

By the end of June, plants suffering from the disease, as described above, may be found; they have not formed any tubers by this time and generally die within a week. In the case of plants which show the disease later in the summer and have already produced tubers, the disease penetrates the tubers and makes the inside black and rotten. Bacteria are present in large numbers in the vascular bundles of the stem as well as in the tissues of the tubers. Tubers from infected plants, if used in the following year for seed, give rise to diseased plants again (Appel, 1903; Morse, 1910; Shapovalov and Edson, 1921). This disease was first noticed in France about 1890, and was then described under the name "Gangrène de la tige." Later it appeared in most of the countries in which potatoes are grown.

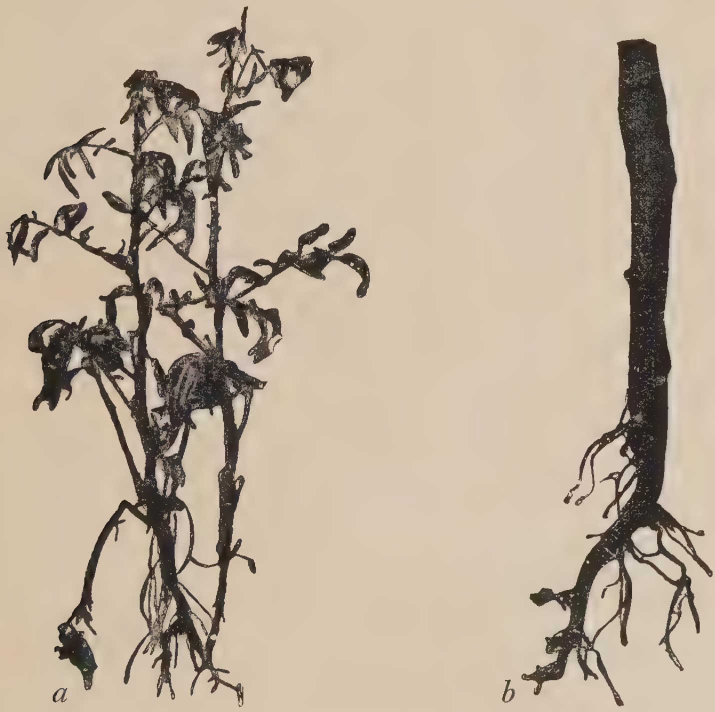

Fig. 4.—Black-Leg of Potato.
a, a diseased plant; *b*, the lower part of a diseased stem.
(*a* after O. Appel; *b*, Orig.)

Control Measures :

(1) Use healthy seed. (2) Plant whole sets. (3) A field on which the disease has appeared in previous years should not be planted with potatoes. (4) Heavy dressings of nitrogenous manures should be avoided. (5) The crop should be examined in summer and any diseased plants removed and destroyed.

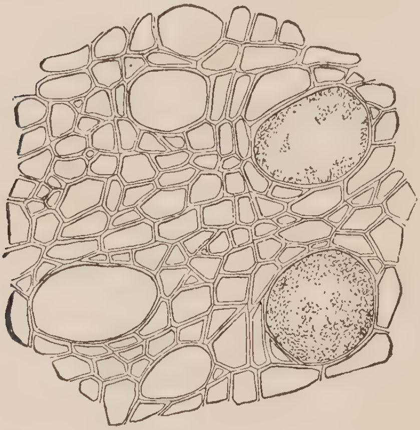

Fig. 5.—Black-Leg of Potato.
Vascular bundle tissues with two vessels containing bacteria, from a stem with disease in lower part. (After O. Appel.)

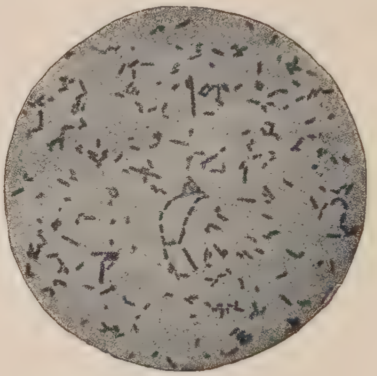

Fig. 6.—*Bacillus phytophthorus.* Highly magnified. (After O. Appel.)

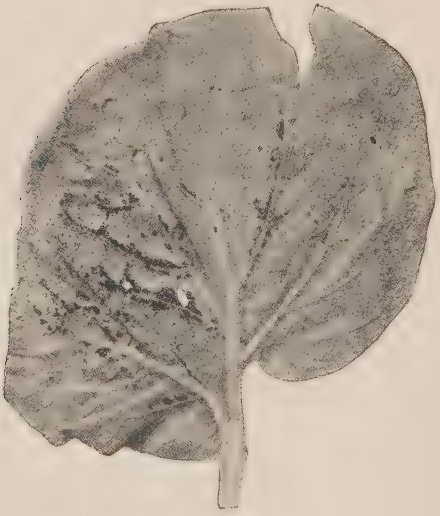

Fig. 7.—Blackening of Veins of Potato Leaf. (Orig.)

Another disease, "Brown Bacteriosis," affecting the stems of potatoes appears later in the summer in France, where it was first observed in 1901; it appeared later in England. The leaves yellow permaturely and the stems become thin and begin to die from below upwards. The fibro-vascular rings of the stalk and in the tubers show yellowish-brown spots with yellow oozing material. The cause of the disease is *Bacillus solanincola* Delacr.

A disease known by the blackening of the main veins of the leaves of potatoes must be regarded as caused by bacteria, although the causal organism has not yet been isolated. The affected leaves quickly die and fall as a black slimy mass to the ground. The disease was observed on early potatoes in Sweden in the years 1910 and 1913.

Bacterial Disease of Mangel and Beet

Bacillus Betæ Mig.

The yellowing and wilting of the fully-developed leaves is the first symptom of this disease of the mangel and sugar beet. On examination the root is found to be blackened at the tip, withered and deformed; the vascular bundles show a reddish-brown discoloration and later become brownish-black. A longitudinal section reveals dark streaks running lengthwise through the root, and in cross-section these are seen as rings. When an infected root is cut a thick, gummy fluid exudes from the browned portions. Finally, the whole of the root becomes brown, the parenchyma disappears, and only the blackened, thread-like vascular bundles remain.

Three forms of bacteria (*Bacillus* α=*B. lacerans* Mig., *Bacillus* β =*B. Bussei* Mig. and *Bacillus* γ) have been isolated from diseased roots, and with one of these forms (β) successful inoculations have been made out of doors.

This disease has been detected in a number of European countries and in many places has caused serious losses.

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Analogous bacterial diseases on sugar beet have been reported from different parts of the United States: one form in Indiana (Arthur and Golden, 1892; Cunningham, 1899), and another in Nebraska, Arizona, and Colorado (Hedgcock and Metcalf, 1902).

In France a disease known as **Dégénérescence Gommeuse** has been found on sugar beet stored in silos. It is considered that a fission fungus closely related to *Bacterium Mori* Boy. & Lamt. is the cause of the disease. Smooth, shining patches appear on the roots, then concentric cavities, and finally the root becomes soft and pulpy. The gum that exudes from the cut surface does not have an unpleasant smell (Arnaud, 1915).

Wart Bacteriosis caused by *Bacterium scabiegenum* Fab. first appears on the surface of the root in the form of small, circular, black warts, often with a crater-like depression in the centre of the wart. On the sugar beet the warts coalesce, whilst on mangels they are generally found on the lower half of the root. The disease does not generally penetrate into the flesh, and in many cases the wounds heal and the warts are set free; severe rotting of the root has not been observed.

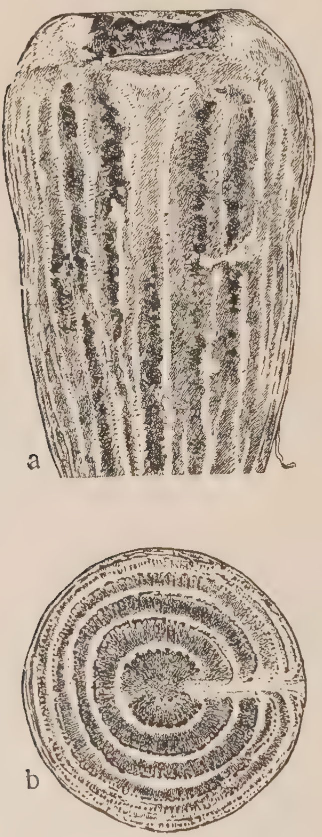

Fig. 8.—Bacterial Disease of Sugar Beet.

a, longitudinal section; *b*. cross-section. (After P. Sorauer.)

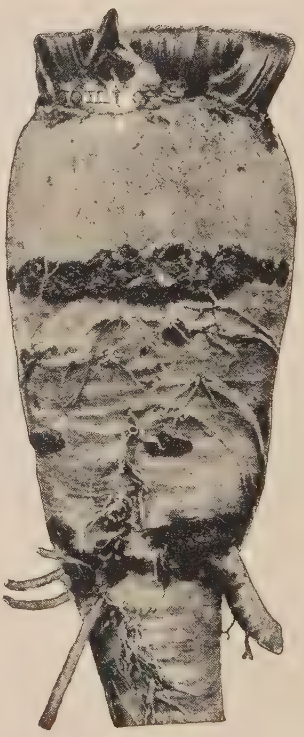

Fig. 9.—Wart Bacteriosis of Sugar Beet. (After F. C. v. Faber.)

The disease has been observed in Germany and in North America (von Faber, 1907).

Californian Bacteriosis has been observed in California since 1899. The infected roots are retarded in development and never become as large as healthy ones. The leaves, which remain small, turn a yellowish-brown and then black; they are often much crinkled. The root-bulbs are sometimes no larger than a radish, and are covered with a felt of numerous small fibrous roots. The interior of the root shows dark, concentric rings. From the vascular bundle a dark-coloured juice, with an extremely bitter taste, oozes out and turns black on exposure to the air. In many cases this disease has destroyed 50–100 per cent. of the crops; it is caused by *Bacillus sp.* Linh. (Linhart, 1901).

Beet Yellows.—This disease first makes its appearance on the outer leaves of sugar beet and mangels and then on the inner leaves. The lamina of the leaf is mottled, bright yellow and dark green, and thus appears as though suffering from mosaic disease. When the leaf is held against the light, this characteristic appearance is more easily seen. The disease spreads from the lamina to the leaf-stalk, which becomes transparent and slimy to the touch. The whole leaf soon dies, the lamina turning to a black, slimy mass. Beets in flower exhibit similar symptoms on the floral organs. In the case of severe attacks of disease the roots fail to develop.

This disease was first observed in the North of France in 1896, and has appeared since 1908 in a few places in Germany and since 1909 in Denmark and Sweden, sometimes very destructively. It has been proved in France that the disease is due to *Bacillus tabificans* Delacr., which is also present on the seed but only remains viable for three years. Infected seed, if kept for this length of time and then sown, will produce healthy plants (Prillieux & Delacroix, 1898; Delacroix, 1908; Eriksson, 1912).

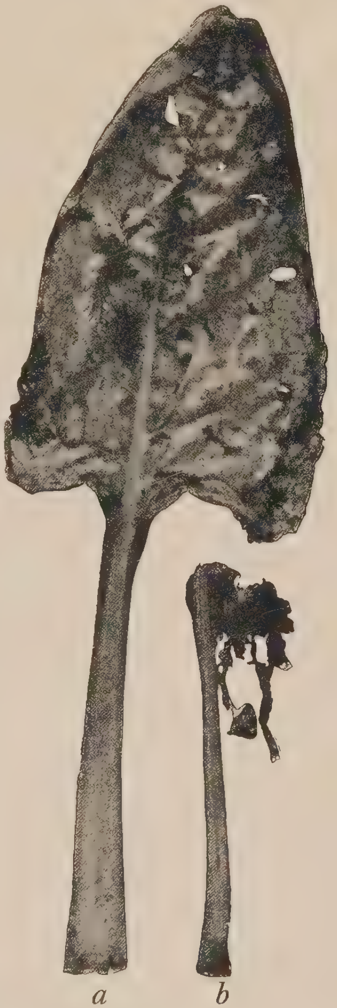

Fig. 10.—Beet Yellows.

a, mosaic appearance of lamina of young leaf; *b*, later stage, lamina withered, leaf-stalk transparent. (Orig.)

A disease known in Denmark as **Mosaic Disease of Mangel** gives rise to changes in the leaf similar to those mentioned above, but in this case the lamina does not wilt and fall nor is the leaf-stalk transparent and slimy. A new crop is not infected through the seed or the soil, and no bacteria have been found to be present in diseased plants. The contagium or virus appears to be present in the cell itself and to be carried—probably by insects—from diseased mother-mangels to young plants in the neighbourhood. The disease first appeared in Denmark in 1899 (Lind, 1915), and it has been known in Sweden since 1910. More recently it appears to have been met with in Moravia (Fallada, 1913) and Northern France (Miège, 1915).

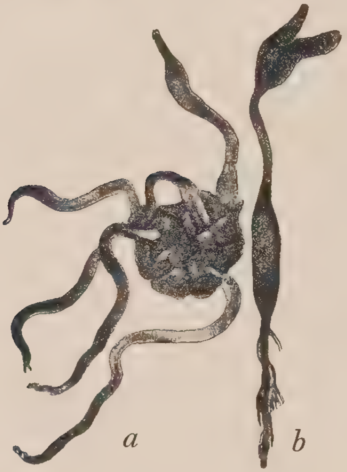

Fig. 11.—Seedling Bacteriosis of Mangel.

a, seed cluster with diseased seedlings ; *b*, diseased seedling. (After G. Linhart.)

Seedling Bacteriosis, *Bacillus mycoides* Linh. attacks the young seedlings of mangel-wurzel at the time when they emerge from the seed and causes a varying amount of damage, in severe cases leading to the death of the seedling. This disease occurs in Hungary mainly (Linhart, 1900).

Black Rot of Crucifers, etc.

Pseudomonas campestris (Pam.) Erw. Sm.

This disease is found on many members of the cabbage family such as the common cabbage, broccoli, cauliflower, kohl-rabi, as well as on swedes, turnips, and rape, sometimes even on radishes and mustard. The plants may be attacked at any stage of growth from the young seedling to the mature plant. If the attack is at an early stage the plant soon dies, or else ceases to grow. In the case of older plants the effect of the disease is often singular ; thus turnips become thin and elongated almost like carrots, whilst cabbages lose their leaves one after another, and the leaf scars on the stem show a number of small black dots.

Infection usually takes place through the leaves, rarely through the roots, and then only at a very early stage. Leaf-eating insects, green fly or slugs, may introduce the disease, or the fungus may enter through the water-pores on the margin of the leaf. It is through these pores that excess of water is transpired, and when the air is warm the moisture evaporates, but after a cool night it collects in drops which may remain for several hours after sunrise. It has been proved that these drops enable the bacteria to gain admittance to the plant, as when the water evaporates any bacteria present in the drops enter the space below the pore and there multiply enormously. The bacteria pass from the cavity to the interior of the leaves, probably by way of the vessels, which in consequence become blackened. Moist, warm weather (about 20° C.) favours the infection to a high degree. It has been possible in a single cabbage plant to trace 400 separate infections by way of the water-pores. The result of infection is that after 10–20 days the leaf takes on a yellowish colour and the veins become black. Under favourable weather conditions the disease may advance 1 cm. per day both downwards into the leaf-stalk, stem, and roots and upwards into the healthy parts of the leaf. In cold weather, 6–8 weeks may elapse before the root is completely rotten.

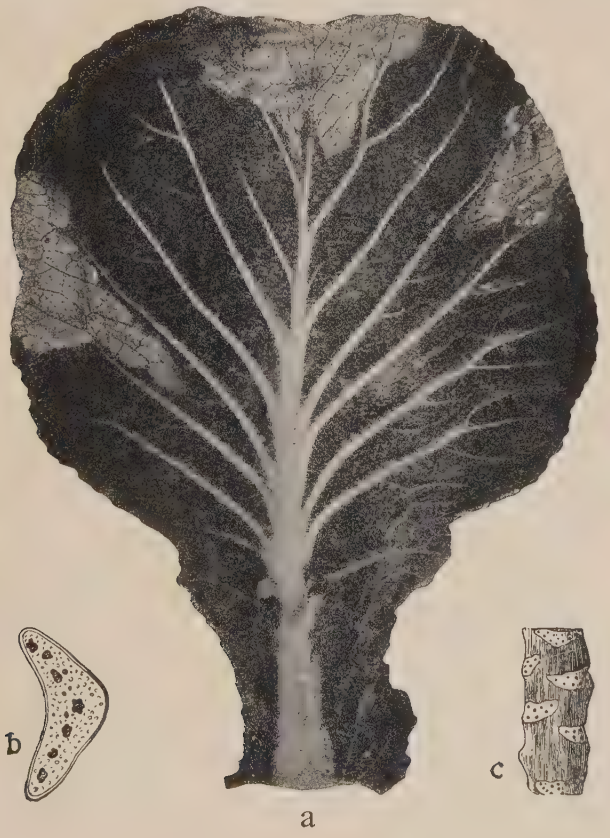

Fig. 12.—Black Rot of Cabbage.

a, cabbage leaf showing three patches on margin due to bacteria; *b*, cross-section of a diseased leaf-stalk with black bundles; *c*, piece of diseased cabbage stem with scars showing black dots. (After Erw. Smith.)

At first, the bacteria are localised in the vascular elements, but they are able to dissolve the thickened walls of the vessels and also the walls of the parenchyma, and in this way cavities of a smaller or larger size are produced.

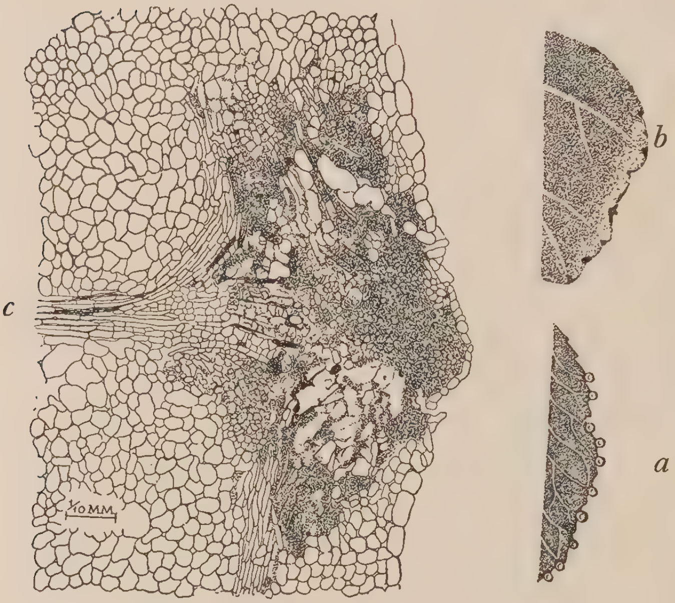

Fig. 13.—Black Rot of Cabbage.

a, margin of leaf with drops of water on the water-pores; *b*, early stage of bacterial infection through water-pores; *c*, cross-section of leaf at point where bacterial infection has taken place through a water-pore. (After Erw. Smith.)

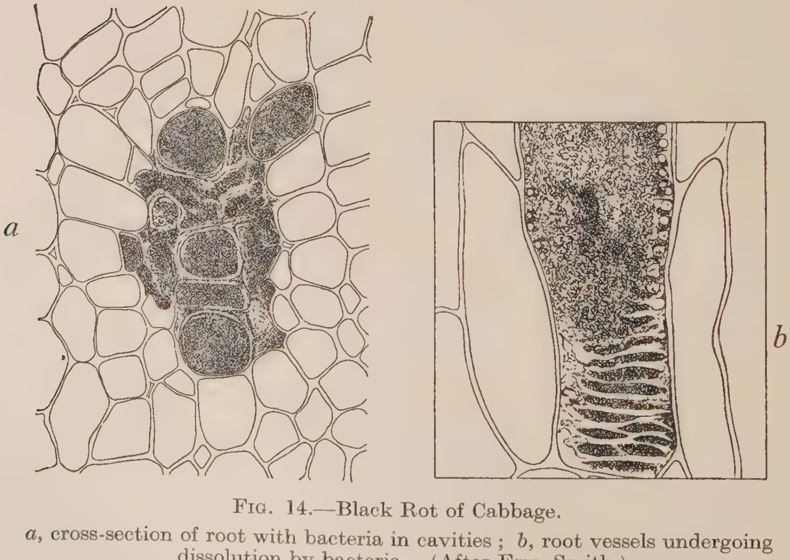

Fig. 14.—Black Rot of Cabbage.

a, cross-section of root with bacteria in cavities; *b*, root vessels undergoing dissolution by bacteria. (After Erw. Smith.)

In the case of turnips the rot takes on a different character and may exhibit two forms; in the one form, longitudinal black streaks are found, and in the other the bundles, which have also been blackened by the bacteria, are arranged radially. In both forms of the disease the turnip shows little alteration externally, but after a time numerous wart-like growths develop on both of the furrows which run the length of the root. In the case of swedes a deep, cross-shaped crack leading to a cavity

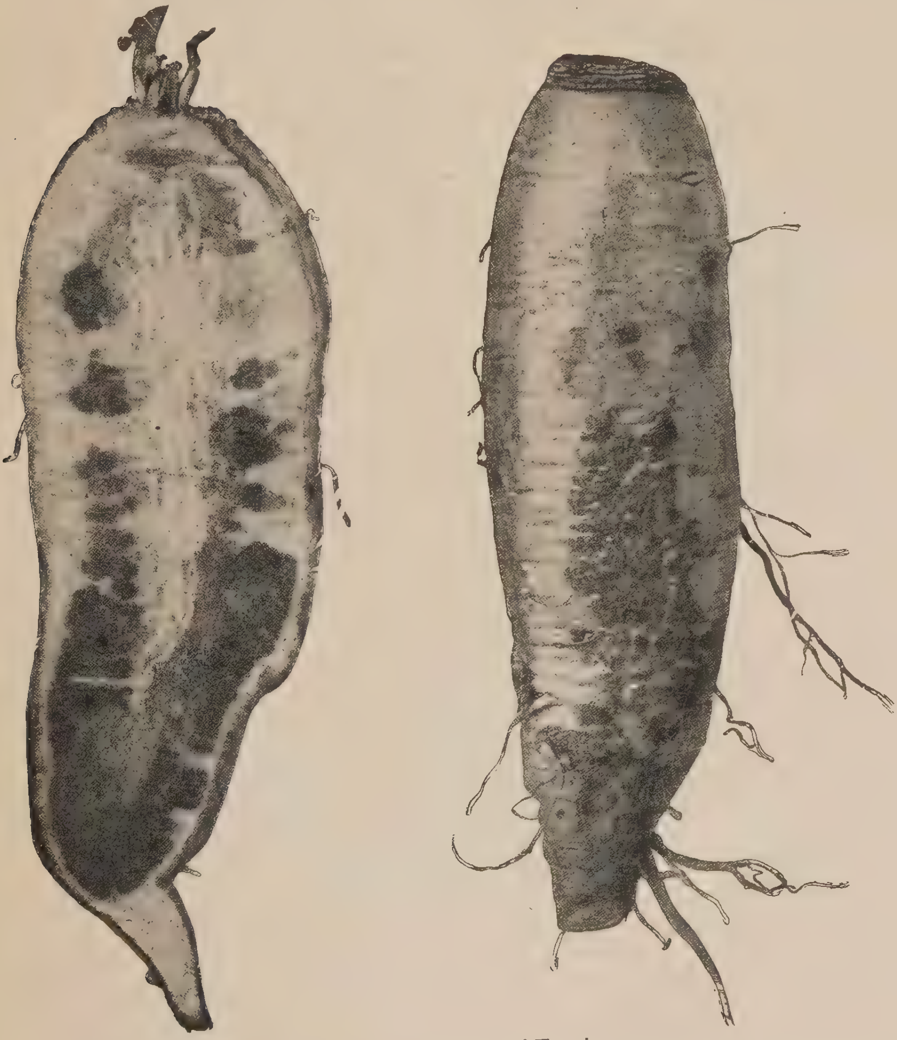

Fig. 15.—Black Rot of Turnip.

a, common bacteriosis: longitudinal section showing two strands of diseased tissue; *b*, wart bacteriosis. (Orig.)

in the root is often characteristic of the disease, whereas in kohl-rabi the bulb is traversed by a network of blackened strands (vessels).

Sometimes a Fusarium disease (*Fusarium Brassicæ* (Thüm) Sacc.) is associated with Black Rot, in which case the roots differ strikingly from the normal in their external appearance. Under the combined attack the roots have an irregular, knotted shape and a rough, dark-coloured skin (Eriksson, 1915).

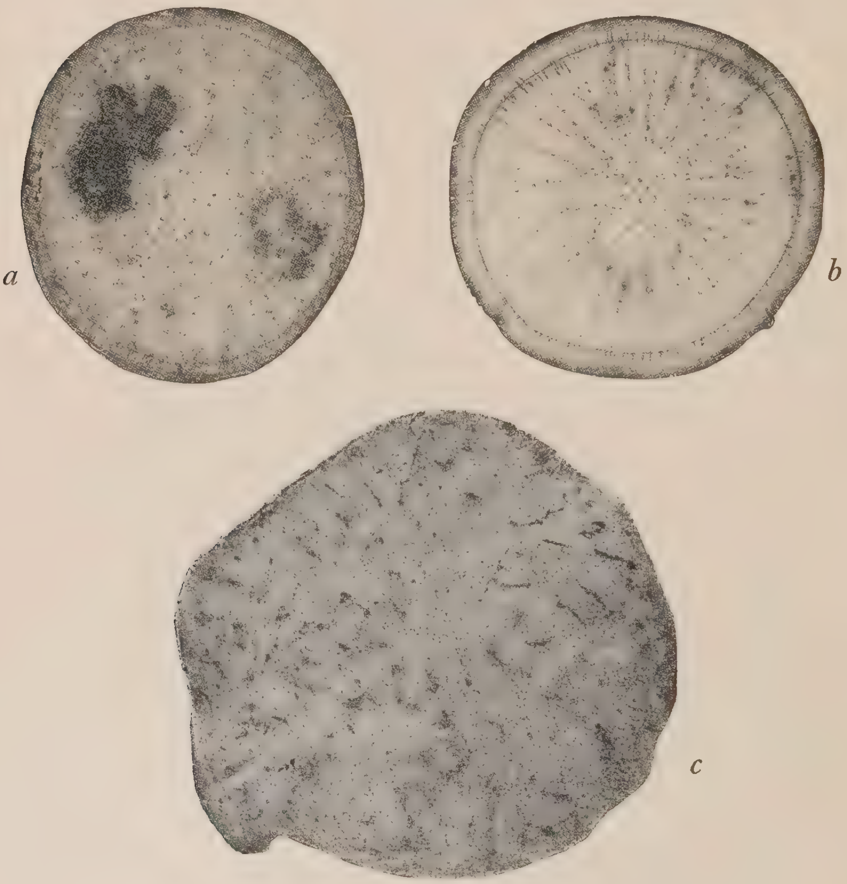

FIG. 16.—Black Rot of Turnip and Kohl-rabi.

a, cross-section of turnip showing longitudinal strands of diseased tissue ; *b*, cross-section of turnip with radial strands of diseased tissue ; *c*, kohl-rabi with network of diseased strands. (*a* and *b*, original ; *c*, after Erw. Smith.)

When crops are stored, the disease spreads rapidly, particularly if the place of storage is warm and damp, and the cabbage, turnips, etc., may be changed by the rot into a slimy, foul-smelling mass.

Seed from diseased plants reproduces the disease (Harding and Stewart, 1904; Clinton, 1912; Walker, 1922, 1924). For this reason it is easy to understand how it is that the disease has spread so rapidly in the last ten years, both in the Old and New World. In the United States it has been estimated that 40–50 per cent. of some crops has been destroyed, and in extreme cases as much as 90 per cent.

Black Rot was first recorded in the United States (Kentucky) in 1890, and it is now widely spread in America. In most of the European countries it has appeared with greater or less virulence during the past decade.

Control Measures :

(1) Seed obtained from infected fields must in no circumstance be used. (2) Young plants raised in seed beds must be carefully examined before planting out, and if any are diseased the whole bed should be destroyed. (3) Cruciferous plants should not be grown for 2–3 years on soil known to be infected with the disease. (4) Dung, particularly if diseased roots have been fed, should not be applied to the seed bed or to the field, and in doubtful cases artificial manures are preferable. (5) In the early stages of the disease when the infection is limited to the margins of the leaf and has not reached the stem, a regular inspection of the field should be made each ten days, and any diseased leaves should be removed and destroyed. If the disease has progressed beyond this stage, then the plants themselves must be destroyed. (6) Diseased parts of plants should not be left lying on the field during the winter. (7) The crop should be stored in a cool, dry, well-ventilated place.

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In addition, the following bacterial diseases have also been observed : **White Rot of Turnips**, which was first found in England in 1900 and later in other countries. The outer, and, later, the inner, leaves of the plant turn yellow and fall to the ground. The disease spreads quickly and within fourteen days the plants may be destroyed. The affected parts of the root become white or greyish-white, never brown or black, and they finally rot. The attack varies with the different kinds of turnips, some being affected up to 23 per cent. of the crop. The disease is caused by *Pseudomonas destructans* Pott. and can pass to other root crops such as the carrot (Potter, 1900). A bacterial disease on cabbage, noticed in Westphalia in 1902 and caused by *Pseudomonas sp.*, has been recorded. This disease starts on the young portions of the stem and on the midrib of the leaves and changes them to a foul-smelling, pasty mass. Often all the plants in a field are destroyed (Spieckermann, 1902).

Two diseases of the cauliflower and other plants of the cabbage family also call for mention. The first, known in Canada since 1901, is due to *Bacillus oleraceæ* Harr. and causes the plants to rot completely to a soft mass (Harrison, 1904). The second, which was first observed in France in 1905, is due to *B. brassicævorus* Delacr. ; the symptoms are the dying off of the terminal buds and the formation of pale patches on the leaves (Delacroix, 1905).

Crown Gall of Mangels occurs here and there on odd plants. The galls are large, tumour-like, and of varying shape ; in size they may be as large as an average potato and of the same colour as the root itself. They have a smooth surface and soon decay.

The galls are the result of infection by *Pseudomonas tumefaciens* Erw. Sm. and Towns., which has the power of stimulating cell division in that part of the root that is infected. The same bacterium—there are probably several biologic forms—can also cause gall formation on other plants such as species of the genera *Chrysanthemum*, *Rosa*, *Medicago*, *Salix*, *Pyrus*, etc. The bacterium has been isolated from thirty-four species of host plants belonging to fourteen families. As a rule, crown gall is not a serious disease of mangels (Townsend, 1915 ; Erw. Smith, 1916, 1917, 1922 ; Brown, 1923 ; Lévine, 1925 ; Riker and Keith, 1925).

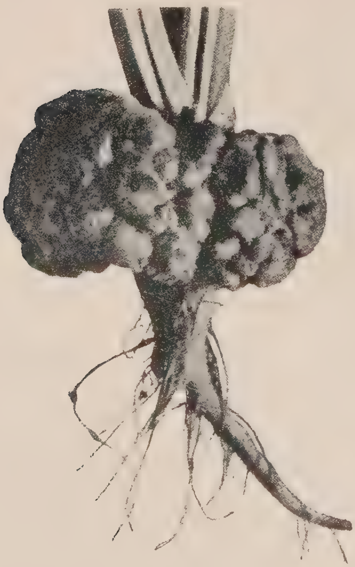

Fig. 17.—Crown Gall of Mangel. (After Erw. Smith.)

A similar formation of galls has been

named **Tuberculosis.** The surface of the galls is rough, cracked, and dark in colour, whilst the internal tissues are of a brownish colour. The disease is caused by *Bacterium beticolum* Erw. Sm.

In certain human and animal diseases (*e.g.* "Meningitis cerebrospinalis"), bacteria have been isolated which, in their morphological, cultural, and serological characters, agree very closely with those of *B. tumefaciens* obtained from crown galls of plants. Artificial inoculation experiments have been undertaken to find out whether the bacteria isolated from plant and animal tumours agree in their behaviour towards plants and animals. The results which have been achieved show that *B. tumefaciens* is both an animal and plant pathogene. It is probable that in the case of the plant a fresh wound on the surface is a necessary condition of successful inoculation, whereas with the animal the bacterium is taken into the body with the food. A possibility thus arises that raw or insufficiently cooked vegetable material containing the organism may produce canker-like growths in the animal body (Friedemann and Magnus, 1915).

Control Measures :

(1) Remove and destroy the plants that are first attacked. (2) Diseased parts of plants must not be left lying on the ground over winter. (3) Care should be taken that none of the soil from an infected field is carried by carts, implements, etc., to a field which is to be used for a root crop in the following year. (4) The remains of diseased plants must not be thrown on to the manure heap. (5) Seed from plants grown on infected soil should not be used. (6) Where there has been an outbreak of disease, roots should not be grown for the next three years.

Bacterial Diseases of Carrot, etc.

The following forms of bacteria are responsible for certain diseases found on agricultural plants : **Bacillus carotovorus** Jon. The carrots when stored develop a wet rot ; the rot usually begins in the crown and spreads downwards, causing the affected parts to become soft and of a brown colour. The disease is also found in other root crops such as turnips, parsnips, radishes, etc. (Jones, 1901, 1909 ; Daumezon, 1919 ; Eisler and Portheim, 1921).—**Bacillus elegans** Heg. appears on lupines. The leaves of the young plants are affected with yellow spots which later turn brown, the leaves soon wither and dry up (Hegyi, 1899).—**Bacillus Cubonianus** Macch. on hemp. Numerous narrow, greyish-white patches (up to 10 cm. in length) on the stems, on the leaves black patches which later destroy the tissue. The organism which causes the above symptoms seems to be identical with the one which gives rise, in France and Italy, to a bacterial disease of the mulberry.

Bacterial Diseases of Cereals and Grasses

"**Red Grain**" appears on barley and sporadically on wheat and rye. The disease manifests itself by brown to brownish-black spots on the base of the leaf-stalk, the nodes, and upper parts of the stem. The upper leaves turn yellow prematurely and die, the ears become notched and jagged, and the grains develop incompletely, often being naked owing to the splitting off of the glumes. Sometimes a gaping crack is found on the back or sides of the grain. The inside of the grain is coloured reddish-brown to brick-red, the red colour being located in the gluten layer. The embryo is often bright red. The disease is prevalent in dry seasons, is carried in the seed, and when corn is stored in a damp place the infection can be transferred to healthy grains.

This disease was first noticed in France in 1878 on wheat, and was ascribed to *Micrococcus Tritici* Prill. It appeared later in other countries, in Denmark on wheat, in Sweden in 1908 on barley, and in Germany, mainly on barley, in 1915–19 (Prillieux, 1879; Gentner, 1920).

In different parts of the United States, especially in the Middle West, another bacterial disease of wheat known as "**Black Chaff of Wheat**" has been observed. Parallel, sunken streaks are first noticed on the glumes, and later the spindle of the ear and the stem become brown or black; yellow or translucent stripes appear on the leaves. In severe cases the grains shrivel and when cut across show small cavities filled with bacteria. The disease is stated to be due to *Bacterium translucens var. undulosum* Erw. Sm. (Jones, Johnson, and Reddy, 1916; Reddy, 1924), and *Bacterium atrofaciens* Cull. (Erw. Smith, 1917; McCulloch, 1920).

Pseudomonas Stewarti Erw. Sm., which causes "Bacterial Wilt of Maize," has been known in the United States of America since 1897. The disease begins about the time of flowering and the first attack is upon the leaves; the plants ultimately wither and become dry. A cross-section through the stem shows a viscid, yellow secretion which oozes from the vascular bundles. Probably the disease is spread by infected seed (Stewart, 1897; Erw. Smith, 1903).

Another disease of maize—"**Corn Blight**"—was observed in the United States about the same time as the previous disease and is stated to be due to *Bacillus Zeæ* Burr. The disease attacks maize plants in various parts of the field, causing them to turn yellow and cease to grow. On the leaf-stalk and on the stem, brown patches appear, also on the roots; the latter contain a slimy substance and soon die. When plants suffering from this disease are fed to cattle a peculiar sickness—Corn Stalk disease—results.

Sorghum Blight is a disease caused by *Bacillus Sorghi* Burr. Yellow patches, which later become red, appear on the leaves and inflorescences; similar patches may almost cover the stem. This disease has been observed in Holland, Italy, and America (Burril, 1889; Bruyning, 1898; Radais, 1899).

In addition, a number of bacterial diseases have been met with on grasses, *e.g.* **Aplanobacter Rathyi** Erw. Sm. on *Dactylis glomerata* (J. Lind, 1917), **Bacterium moniliformans** Griler, on the swollen nodes of the rhizomes of *Arrhenatherum elatius*, etc.

Bacterial Diseases of Tobacco

There are a number of these diseases; one of them, known as **White Rust,** causes small, irregular patches on the leaves. These patches are green, then white, and are bounded by a layer of corky tissue. The patches and the bacteria within finally dry up. The disease is attributed to *Bacillus maculicola* Delacr. (Delacroix, 1905). Another disease, termed **Anthracnose,** produces long, deep, dark patches on the stems and on the midrib of the leaf and may lead to the death of the plant. This disease, which is very common in France, is stated to be due to *Bacillus æruginosus* Delacr. and *B. tabacivorus* Delacr. (Delacroix, 1906).

In recent years, a number of new bacterial diseases have been reported from the United States. One of these, "**Wild Fire,**" attacks the leaves of plants in the seed bed and those in the field. Round, yellow patches, as large as the tip of a finger, in the middle of which is a small white-brown spot. This spot extends until only a very small part of the yellow remains visible. When the attack of the disease is severe a considerable portion of the leaf tissue is killed. This disease was first observed in North Carolina in 1917 and it has now spread to at least forty of the American states and is the cause of much loss. It is due to *Bacterium tabaficum* Wolf & Forst. (Clinton, 1922; Johnson and Fracker, 1922; Thomas, 1924; Anderson, P. J., 1925). Closely allied to the above disease are "**Black Fire,**" caused by *Bacterium angulatum* From. (Fromme and Wingard), and "**Yellow Rust,**" caused by *Bacterium melleum* Johns. (Johnson, 1923).

BACTERIAL DISEASES OF VEGETABLES

Bacterial Blight of Celery

Pseudomonas Apii Jagg.

This disease gives rise to small, rust-red, irregularly-shaped spots which gradually enlarge and may finally coalesce: in damp weather the patches usually have a wet, shining appearance. The first traces of the disease are seen in the seed bed, particularly in years when the rainfall in May and June is heavy. It is not known whether the disease can be spread through the seed, but there are indications that it can. Secondary infection in the field is rapid in wet weather. The bacteria causing the disease are found in large masses in the leaf spots.

The disease has been very destructive in America (New York State) since 1910. The fungus was described in 1921 under the name of *Pseudomonas Apii* Jagg., and in 1924 copper-containing fungicides (both liquid and powder) were used against it. It is recommended that seed beds should be sprayed several times—up to four—with fungicides, starting when the seedlings develop the first two leaves and continuing up to the time they are planted out.

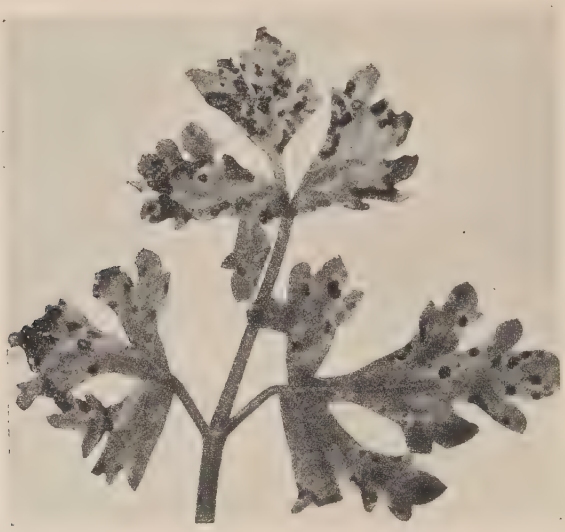

FIG. 18.—Bacterial Disease of Celery.
(After H. W. Dye and A. G. Newhall.)

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A bacterial disease of celery, previously observed in Italy (Brizi, 1897), is probably identical with the above. In America (Florida) another celery disease characterized by the yellowing and drying up of the tips and edges of the heart leaves has been noted (Winters, 1908).

Crown Rot of Rhubarb is a disease caused by *Bacterium rhaponticum* Mill. The upper parts of the root become rotten, the sheaths of the new buds darken, and the base of the leaf-stalk swells considerably; the lamina of the leaf turns dark brown. Affected plants cannot be cured; they must be dug up and the soil disinfected with a 3–5 per cent. solution of ammonia. In the years 1915–19 the disease was very severe in Yorkshire (England) and caused heavy losses (Millard, 1924).

Bacterial Diseases of Lettuce.—At the beginning of this century a bacterial disease was found in Italy on *Lactuca sativa*. The plants showed a softening of the tissues of the leaf and stem; in severe cases the tissues became brown and mouldy. In the diseased leaf tissues, accumulations of *Bacillus Lactucæ* Vogl. were found. As a result of this disease the growing of lettuce in the neighbourhood of Turin was reduced to a fifth of the normal supply (Voglino, 1903). Recently another bacterial disease has been observed in England. The leaves turn brown, generally at the edges first, and the disease spreads inwards to the midrib, forming a brown network. The organism is *Bacterium aptatum* Pain. & Bran. In 1921 this disease destroyed 75 per cent. of a lettuce crop (Paine and Branfoot, 1924).

In America, numerous other bacterial diseases of lettuce are known; one which appeared in Florida causes the margins of the leaf to become dry and black and finally the whole leaf rots (Rolfs-Burger, 1914). Another disease which caused much damage in 1915 in Louisiana was characterized by watery-looking patches on the leaves, and soon afterwards most of the latter died. Large colonies of *Bacterium viridilividum* Br. (Brown, 1915) were found between the cells.

BACTERIAL DISEASES OF TOMATO

Blossom-end Rot

Phytobacter lycopersicum Groen.

Young tomatoes attacked by this disease turn brown at the blossom end of the fruit, and from this point the disease spreads centrifugally to other parts. The flesh ultimately decays and the fruit becomes inedible. The disease has been known since 1895 in France, 1896 in America, and 1902 in Denmark. In 1922 the disease was met with in Holland and Astrachan, and in 1924 in the Dutch Indies (van Hall, 1924).

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Tomato Rot is a disease similar to the foregoing and was first noted in Northern Russia. Dark brown patches appear on the fruit near the style and quickly spread until the whole of the end of the fruit is affected. As the fruit ripens the patches become sunken, shortly afterwards the fruit drops and is found to be filled with a dirty, pasty mass having an ammonical scent. A pure culture of the organism causing the disease leads to the view that it is *Bacterium Lycopersici* Burgw. A large number of tomato varieties appear to be susceptible to the disease, which in some cases may destroy the whole crop. The suggested measures of control are: (1) Use seed from healthy plants, or if this cannot be assured the seed should be disinfected for five minutes with 10 per cent. formaldehyde or 1 : 4000 corrosive sublimate solutions. (2) Keep the soil moderately moist. (3) Avoid excessive manuring with nitrogen. (4) Burn all diseased plants at once (Burgwitz, 1924).

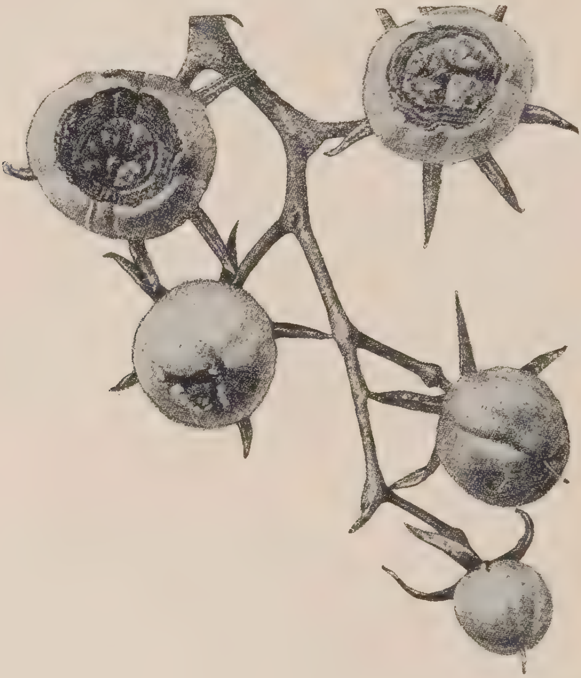

FIG. 19.—Blossom-end Rot of Tomato. (After E. Prillieux.)

In the United States (Indiana) and in South Africa (Transvaal) another disease, "**Bacterial Spot**" (due to *Bacterium vesicatorium* Doidge), has been noticed. Circular, sunken spots, black in the centre and surrounded by a white halo, are distributed over the whole surface of the fruit. Many different sorts of tomatoes were found to be susceptible to the disease, which can also be transferred to species of *Solanum, Datura, Physalis, Lycium, Nicotiana*, etc. The fungus over-winters in the soil, and was found to be still viable on seed kept dry for 16½ months (Gardener and Kendrick, 1923). From America (Virginia) "**Bacterial Soft Rot**" of tomatoes has been reported as another serious disease. A sharply defined, sunken patch appears on the unripe fruit; the surface of this patch becomes wrinkled and cracked and within 3–10 days the whole fruit is changed to a colourless, watery, rotten mass. The decayed fruit finally bursts and the contents is poured out. All the varieties of tomatoes that have been tested have shown themselves to be equally susceptible to the disease, the cause of which is given as *Bacillus Aroideæ* Towns. (Townsend, 1904; Wingard, 1924).

In addition to the bacterial diseases of the fruit, there are others which attack the foliage of the tomato. One of these diseases, **Bacterial Wilt**, manifests itself by a sudden wilting of the leaves on one branch, and this condition gradually spreads to the whole plant. In young plants the stem shrivels, first turning yellow and then brown to black. The vascular bundles of diseased stems are of a dark colour and in severe cases the pith is changed into a foul-smelling, slimy mass. In the end the stem with the dry leaves falls over. The fungus is believed to be *Bacillus Solanacearum* Erw. Sm., the same species of bacterium as that which causes Bacterial Wilt of potatoes" (see p. 6).

This wilt disease of tomatoes has been very destructive in Holland, Italy, and Switzerland and in several districts in America (Mississippi, Alabama, Florida). In America the disease has also appeared on the potato and *Solanum Melongena*. Another wilt disease of the foliage has been observed in Italy (Chieti). The leaves wilt and dry, the stem becomes flaccid, and the plant finally dies. The causative fungus is *Aplanobacter michiganensis* Erw. Sm. This disease is known in America as "**Grand Rapids Disease,**" and has there caused a great deal of damage (Peglion, 1915; Paine and Lacey, 1922). "**Stripe Disease**" is closely related to, if not identical with, the one just described; it is caused by *Bacillus Lathyri*, Manns. & Taub. (Paine and Bewley, 1923).

A new foliage disease of tomatoes appeared at the Cheshunt Research Station (England) in May, 1924. The head of the plant fell over and the inside of the stem was found to be decayed. The disease was caused by *Bacillus carotovorus* Jon. (Butcher, 1925) (see p. 21).

Bacterial Disease of Spanish Pepper.—In 1912 a disease on *Capsicum annuum* was observed in Northern Italy (Bergamo). The causal fungus is *Bacillus Capisici* Pav. & Torr., and the symptoms are an irregular spotting of the stems, branches, leaf, and fruit shoots (Pavarino and Turconi, 1913).

Angular Leaf-Spot of Cucumber

Bacterium lachrymans Erw. Sm. and Bryan

This disease appears on the leaves of cucumber plants (*Cucumis sativus, C. anguria, C. acutangulis, C. dipsaceus, Lagenaria vulgaris,* etc.) in the form of brown, angular spots which at first are separate and sharply outlined, but later coalesce. In the early stages, viscous drops, containing large numbers of bacteria, are formed on the under side of the leaf, and these drops dry up in a few days. The leaf assumes a white to yellowish-brown colour and the surface has a roughened appearance; similar spots and drops of liquid are found on the stems. The spots on the fructifications are few in number and only superficial, but the lesions produced on the epidermis allow of other parasites gaining entrance. Continued wet weather favours the disease to a high degree and leads finally to the death of the plant. In dry weather, the bacteria sometimes die off and the plants recover to some extent, but the crop is reduced.

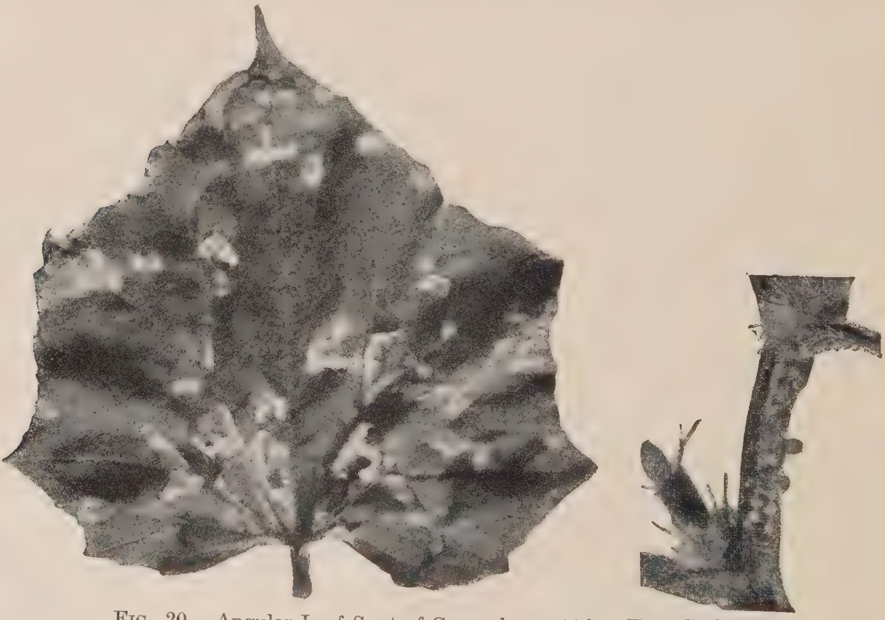

Fig. 20.—Angular Leaf-Spot of Cucumber. (After Erw. Smith.)

The bacteria live in the soil from year to year and they can also be disseminated on seed from a diseased crop. In such cases circular or angular spots having a water-soaked, translucent appearance often appear on the cotyledons of the seedlings. Insects almost certainly play a part in the spread of the disease.

The leaves are infected by day through the stomata, without the epidermis requiring to be injured; during the night the stomata are closed. Agglomerations of bacteria are formed in the intercellular spaces.

This disease was well known to American cucumber growers at the end of the last century. It was much later that it was investigated and the cause shown to be *Bacterium lacrymans* Erw. Sm. and Bry. About the same time the disease was found in Italy (Traverso, 1915) and Russia (Potebnia, 1915–16). As a preventive measure it is advised to steep the seed in a solution of mercuric chloride (1 : 1000) for five minutes (Milbrath, 1922 ; Meier and Link, 1922).

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Another disease of cucumbers—**Cucumber Wilt**—which attacks species of the genera *Cucumis*, *Melo*, and *Cucurbita*, has been known in North America for about the same time as the Angular Leaf-Spot disease, but was investigated at an earlier date. The symptoms of the disease are a wilting and crumpling of the leaves, the stems remaining green and turgid. The stem is, in fact, also affected, the vessels, and, later, the tracheides, being filled with a viscid mass of bacteria which have passed into them from the leaves. Finally, all the tissues are more or less destroyed, cavities being formed which are filled by bacteria. Only the epidermis remains unchanged and the stem, up to the final wilting, remains dry. The cause of the disease is *Bacillus tracheifilus* Erw. Sm. In America this is the most serious of all cucumber diseases and is widely spread (Erw. Smith, 1895, 1896, 1911). The disease has also been found in Russia (Iwanoff, 1898).

Soft Rot of Onion

Bacillus cepivorus Delacr.

In wet years, the outer, hard scales of stored onions are sometimes found to be covered with a grey. floccose mould. The softer scales below show patches of translucent, decayed, foul-smelling tissue in which bacteria are present in large quantities.

From such foci the rot quickly spreads in all directions, attacking the hard, outermost scales and converting them into a soft, ulcerous mass. A characteristic feature of the disease at this stage is the dark margin surrounding the pale centres of infection on the scales.

Onions similarly diseased may also occur in the unharvested crop, in which case earth is found adhering to the decayed part of the bulb.

When the earth is removed the diseased parts are seen to be shrunken and wet. When such an onion is exposed to the air the neck is soon covered with flocculent growths of *Fusisporium*, *Hypomyces*, etc. Continued dull weather with frequent rain and a high temperature favour in the highest degree the increase and activity of the bacteria. If, on the other hand, a dry period of sufficient length supervenes the rot is checked and the onion throws off the diseased portions.

The composition of the bacterial flora in the lesions changes in different stages of development of the rot. When the whole patch is converted into a grey, slimy mass the bacteria are mainly delicate motile rods. In France the disease is considered to be mainly due to *Bacillus cepivorus* Delacr. (Delacroix and Maublanc, 1902).

To decide the part played by bacteria and fungi respectively in producing the disease, various inoculation experiments have been made. The results show that bacteria alone cause the disease, and consequently the fungi must be regarded as entering at a later period ; perhaps they assist (by means of the mycelium) in the spread of the bacterial colonies within the host. This disease was first described from Germany, and later a similar disease, to which the name " Soft Rot " was given, appeared in the Eastern States of North America, causing much damage (Stewart, 1899).

As a preventive measure the soil should be properly drained and later the place in which the crop is stored should be well ventilated.

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Bacterial Disease of Beans.—This disease attacks a number of varieties of beans (*Phaseolus vulgaris*), giving rise to irregular, somewhat sunken, water-soaked lesions on the stem, leaves, and especially the pods. The patches on the pods may penetrate to the young, developing seeds and infect them also. In dry weather the patches become brown and dry up. The cause of the disease is given as *Bacillus Phaseoli* Erw. Sm. and large numbers of bacteria are dispersed through the vessels of the stem and root. Living bacteria have been found on beans 2–3 years old and plants grown from such seed develop the disease. All the varieties of beans that have been tested have shown themselves more or less susceptible to the disease. Of 72 sorts grown in American comparative trials in 1921, it was found that 15 were slightly attacked, 31 moderately, and 26 very heavily ; the degree of resistance of each variety seems to remain constant for several years. The disease is known in many European countries and in the chief bean-growing districts of the United States, where it causes heavy losses. By inoculation experiments the disease has been transmitted to other species of beans such as *Phaseolus lunatus*, *Ph. acutifolius* var. *latifolius*, *Ph. aconitifolius*, *Ph. angularis*, and *Ph. aureus*, also to *Vigna sinensis*, *Soya max*, and *Stizolobium deeringeanum* (Delacroix, 1899; Burkholder, 1921, 1924.)

In the United States a disease of soya bean (*Glycine hispida*) has been distinguished and named "**Bacterial Pustule.**" On one side or both of the leaf, wart-like pustules appear, these are light green, later reddish-brown, and surrounded by a watery halo. The disease is caused by *Bacillus Phaseoli soyensis* Hedg. and has been found in Texas, Virginia, Louisiana, South Carolina, Kansas, Delaware, and Arkansas (Hedges, 1925; Wolf, 1925).

In addition to the above, a special bacterial disease of *Phaseolus lunatus* has been found in America (Wisconsin). Irregular, dry patches, which coalesce gradually and have a red margin and a grey centre, appear on the leaves, whilst reddish-brown streaks are found on the leaf-stalk. On the pods there are small brown spots with a watery, halo-like margin. The disease is stated to be due to *Bacterium viridifaciens* Tisd. & Will., and the bacteria appear to over-winter on the seeds (Tisdale and Williamson, 1923).

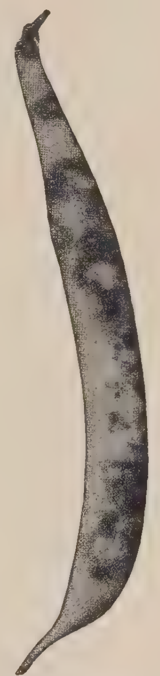
FIG. 21.—Bacterial Disease of Bean. (Orig.)

BACTERIAL DISEASES OF FRUIT TREES, ETC.

Bacterial Canker of Apple and Pear Trees

Bacterium Mali Brzez. and *Bacterium Piri* Brzez.

A canker disease of apple and pear trees, known in Galicia since the beginning of this century, has been shown to be due to bacteria. In external characters this disease is very similar to the *Nectria* canker which is described later in this volume. Bacterial canker appears as wide, cracked patches or open wounds (open canker), or as knot-like, closed swellings (closed canker) on the smaller branches, or as small nodules, of the size of a pea or nut, on the roots (root galls).

When cut lengthwise, the inside of the cankered parts is seen to contain long, yellow to brown strands of tissue which generally start from a canker wound and can be traced upwards for a distance of 40–50 cm. A microscopical examination of the discoloured strands shows colonies of bacteria to be present in enormous numbers. The bacteria are found in all living cells, particularly in the parenchyma of the wood and cortex; they collect near to starch grains, which they quickly bring into solution. Careful investigations extending over a number of years have proved that the bacteria are the true primary cause of the disease and that they are able to induce it in healthy trees.

There are two forms in which bacterial canker manifests itself:

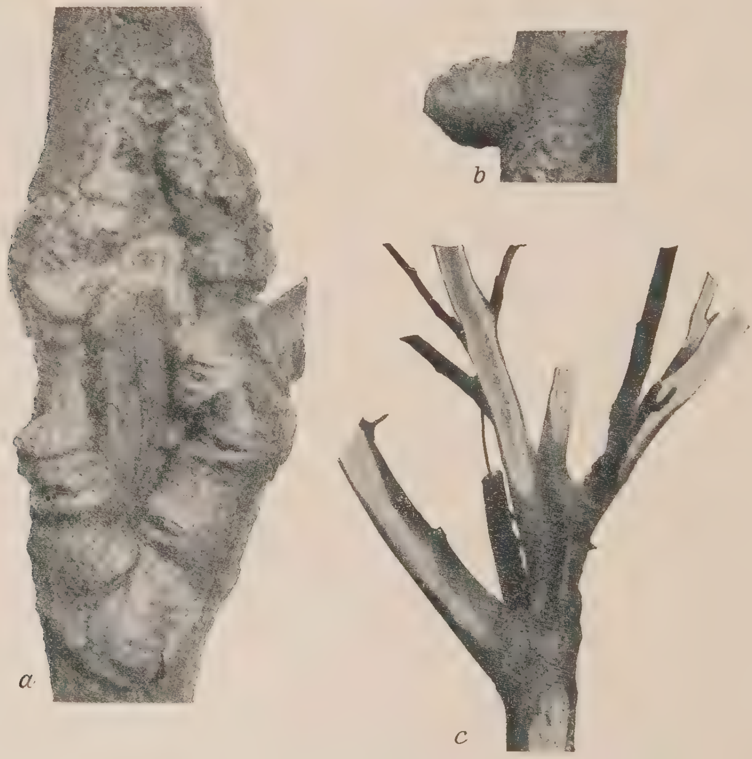

Fig. 22.—Bacterial Canker of Apple Tree.
a, open canker on stem; *b*, closed canker on twig; *c*, cut stem showing strands containing bacteria. (After B. Brzezinski.)

(*a*) the acute form in which the canker spreads rapidly, the bark dries up, and the cambium dies without callus being formed; (*b*) a general bacteriosis in which growth is retarded, a few pale sickly leaves only are produced, and the small twigs die off without apparent cause. Two biologically specialised forms of the bacterium causing canker are distinguished: (1) f. sp. Mali=*Bacterium Mali* Brzez. on *Pyrus Malus*; and (2) f. sp. Piri=*Bacterium Piri* Brzez. on *Pyrus communis*.

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Very closely related to the above disease is **Bacterial Canker of Hazel**, which has been observed in Galicia. This disease is caused by *Bacterium Coryli* Brzez. which attacks *Corylus Avellana* and *C. Colurna*, producing similar cankers.

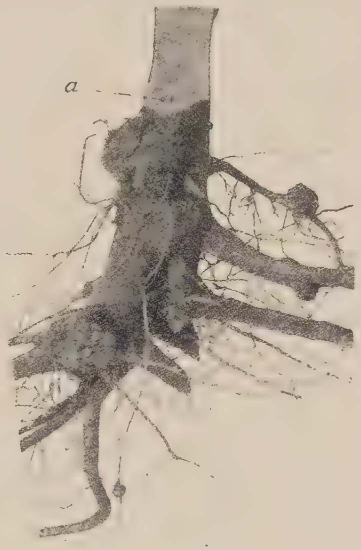

Fig. 23.—Root of Three-year-old Apple Tree with Canker Galls. *a*, place of entrance of bacteria. (After Brzezinski, 1903.)

Some investigators were at one time opposed to the conclusions drawn from the above observations (Aderhold, 1903, 1904), but the possibility of a canker of bacterial nature occurring on the trees mentioned above appears now to be accepted (Aderhold, 1906). Compare Apple Canker, p. 315.

Bacterial Blight of Apple and Pear.—A disease known in the United States as "**Fire Blight**" or "**Pear Blight**" attacks pear, quince, and apple trees growing in the nursery, as well as crab apple, species of *Cratægus* and *Sorbus*, *Pyrus americana*, *Amelanchier canadensis*, *Eriobotrica japonica* and *Heteromeles arbutifolia*; it has also been found on two stone fruits, *Prunus americana nigra* and *P. triloba*. The disease is caused by *Bacillus amylivorus* (Burr.) Trev.

In the nursery, the first signs of disease are seen on two-year-old quince plants, and in the orchard on pear and apple trees in blossom. The flowers suddenly turn brown or black and droop, as do also the adjacent young leaves (Blossom Blight). Soon a similar condition affects the young twigs which become black and die off (Twig Blight). The young developing fruits, when present, dry up and die (Fruit Blight), finally the whole head of the tree has the appearance of having been injured by fire. The disease spreads in the older branches and stem, causing a gummy exudation. The bacteria are found inside the diseased organs, partly in the intercellular spaces, partly in the cells themselves. They hibernate in centres (Hold-over cankers) in the older branches or in the tissues of the bark adjoining a focus of disease in the twigs. When the temperature rises in the spring and the sap ascends, the bacteria become active, multiply and spread in various directions. They give rise to small warts or wounds on the surface of the infected parts, and from these lesions bacteria exude in enormous numbers and are carried by insects (aphides, flies, etc.) to the blossoms and tender leaves of other trees. Injuries on the leaves admit of the entrance of the bacteria, which then pass into the tissues of the twigs and branches.

The disease was first noticed in 1780 in a nursery near to New York (Hudson River Highlands). From this point it has spread widely, being found now in all fruit-growing districts of the United States, and in some years it has been particularly severe. Serious attacks do not, generally, occur in consecutive seasons but at intervals of 5, 10, or 15 years (Burril, 1878–84 ; Arthur, 1885–87 ; Waite, 1898 ; D. H. Jones, 1909 ; Day, 1924 ; Brooks, 1926). The various sorts of apples and pears differ in their susceptibility to the disease (Swingle, 1922 ; Brooks, 1926).

The disease appears, lately, to have gained a foothold in certain parts of Europe, in Switzerland in 1912 (Osterwalder, 1915), and again in 1921–23 in two places, also on pears (Müller-Thurgau and Osterwalder, 1924). A similar disease was noticed 1913–15 in Baden on pear trees (v. Wahl, 1916), and in Italy on apricots (Savastano, 1923).

In many parts of the United States the disease has been considerably reduced by the adoption of the following measures : (1) the careful removal of the bacterial centres (Hold-over cankers) by cutting away the outer bark nearly to the cambium layer and painting the wounds with a fungicide ; (2) the destruction of the blossoms and the young adjoining leaves of all the quince plants in the nurseries ; and (3) by the repeated clearing, in summer, of all centres of infection from nurseries and gardens. (Whetzel and Stewart, 1909 ; Stewart, 1913.)

Since the white thorn (*Cratægus*) has been shown to be responsible for the distribution of the disease, the removal of these bushes has been made compulsory in New Zealand since 1921–22 (Cockayne, 1921).

Bacterial Disease of Cherry Tree

Bacillus spongiosus Aderh. and Ruhl.

This disease has been found on two-year-old wild cherries or on three- to five-year-old grafted trees, and it makes its appearance about the middle of May. The newly-developed shoots suddenly die off with their leaves and flowers ; the twigs cease to make fresh shoots and within a short time, often by the end of June, the head of the tree is more or less withered. It is the trees in the nursery which are mainly attacked, sometimes to the extent of 40–50 per cent. In young trees the disease generally spreads to the stem and descends to the roots, causing the tree to die. In other cases the stem remains healthy and throws up numerous water sprouts, or a branch only may be attacked, the early fall of the leaves indicating the disease. By artificial inoculation experiments it has been possible to follow the course of the disease inside the twigs. Sometimes the bark was killed upwards to the tip, sometimes downwards to the stem, or, again, in both directions, whilst finally the course of the disease was laterally in one or more directions. Where the patches of rot occur, the bark is sunken, brown, and often saturated with gum. This gum is a turbid, pus-like mass, yellowish-green at first and later dark reddish-brown, which exudes in drops from the bark. The twigs may, however, die without the formation of gum.

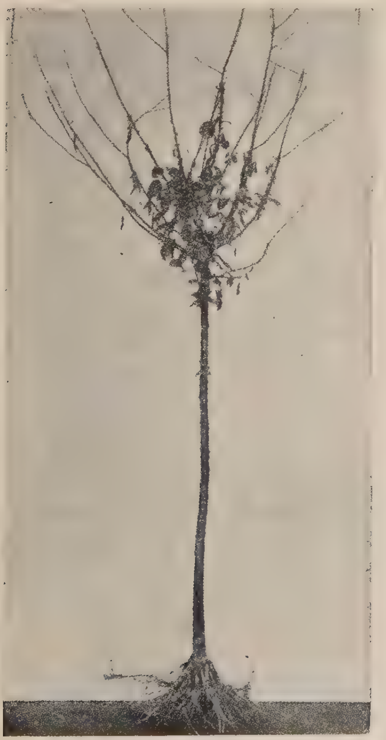

Fig. 24.—Bacterial Disease of Cherry Tree. (After R. Aderhold and W. Ruhland.)

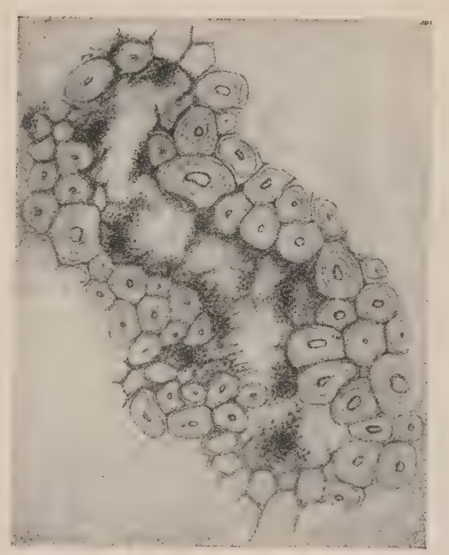

Fig. 25.—Bacterial Disease of Cherry Tree. (After R. Aderhold and W. Ruhland.)

Very large numbers of bacteria are present in the cavities of the tissues which are found on the young bark of the diseased twigs and in the smaller cavities, which arise from the more resistant medullary rays. It has been proved by isolation and inoculation experiments that a bacterium—*Bacillus spongiosus*—present in these foci is the cause of the disease. Another organism, *Bacillus irritans* Aderh. & Ruhl., which is of spherical or coccus form, is generally found, together with the rod-shaped *Bacillus spongiosus*.

The disease appeared in several places in Germany in 1905, in Holland in 1922 (van Poeteren, 1923), and in Switzerland in 1921–23 (Müller-Thurgau and Osterwalder, 1924).

It is not certain if this disease also attacks plum, peach, and apple trees, but apparently it is not identical with the well-known "Fire Blight" of pears (see p. 33). The disease must not be confused with one which was observed in 1903 in St. Goar, where cherry trees died as the result of infection by an Ascomycetes fungus, *Valsa leucostoma* (Pers.) Fr. (see p. 373).

Control Measures :

(1) The centres of infection on the twigs should be cut out, down to healthy tissue, and the wounds painted with coal tar. (2) In severe cases the diseased twigs and branches, or the whole tree, must be removed and burnt. (3) The tools used in infected orchards must be carefully disinfected.

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Clostridium Persicæ-tubercularis Cav. attacks the peach tree, giving rise to nodular growths on the one- and two-year-old twigs, but rarely on the older ones. The bacteria responsible for the disease are found in the infected tissues. This disease was first observed at Pavia in Italy (Cavara, 1897). **Pseudomonas Pruni** Erw. Sm. produces small water-soaked spots on the young leaves of *Prunus japonicus*, and these spots after a time give place to holes. The disease also produces large, dark purple patches on the green fruits, one or two on each fruit ; later these patches change into sunken fissures. Within the stomata, and also in the deeper tissues, large aggregations of bacteria, which have entered the stomata through insect agency, are found. The tissues of the parenchyma are first destroyed and afterwards the vascular bundles. Often the masses of bacteria ooze from the browned surface of the leaves in the form of pale yellow, viscous drops (Erw. Smith, 1903, 1905, 1911).

The same organism—often named *Bacterium Pruni*—is given as the cause of **"Bacterial Shot-Hole,"** a disease much discussed in recent American literature and occurring on stone fruits. This disease attacks peaches, plums, cherries, and apricots, and in various districts in North America (Delaware, Ohio, Illinois) causes much damage, sometimes 50–70 per cent. of the trees being defoliated (Adams, 1923, 1924, 1925; Orton and Wood, 1924).

Bacterial Disease of Walnut Tree.—The disease appears on the tender twigs, leaves, and fruit. The twigs are attacked at the tips and the leaves usually along the veins. Sunken black spots appear on the nuts, the shell becomes soft, the kernel black, decayed, and inedible. The cause of the disease, *Pseudomonas Juglandis* Pierce, over-winters in the fruit or in the cavities of the pith. In South California the disease was so serious that a prize of 20,000 dollars was offered for a remedy (Pierce, 1901; Güssow, 1911; C. O. Smith, 1921). Lately the disease has appeared with devastating results in South Australia (Osborn and Samuel, 1922) and New Zealand (1923) and appears to be invading Europe, having been noted in Holland (van Poeteren, 1923), Italy (Savastano, 1923), Switzerland (Müller-Thurgau and Osterwalder, 1924).

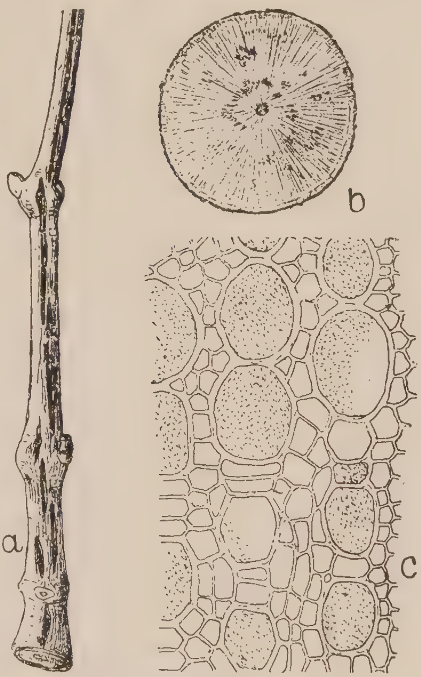

Fig. 26.—" Mal Nero " of Vine.
a, piece of branch with lesions; *b*, cross-section of stem; *c*, cell tissue with bacteria. (After P. Ferraris.)

"Mal nero" of the vine. This well-known disease has spread widely and has been very destructive. Brownish-black streaks on the stem of the vine are characteristic, but the disease is generally first noticed owing to the stunted growth of the tendrils, leaves, and blossoms. A cross-section of a diseased twig shows black dots and lines which run longitudinally and radially. Within the vessels and the cells of the black portions of the stem, large numbers of bacteria are congregated. Various names have been assigned to the bacteria, *Bacillus vitivorus* Bacc., *Bacterium gummis* Cug., *Bacillus Baccarinii* Macch. The colonies of bacteria, and with them the symptoms of disease, spread downwards through the whole stem, the streaks on the stem increase until frequently deep cracks penetrating to the medullary tissue are produced. After about five years the vine is usually killed unless suitable protective measures are taken in time.

Views differ as to the part played by bacteria in the disease. Italian investigators regard the bacteria as the primary cause (Comes, 1884 ; Cugini, 1892 ; Baccarini, 1894 ; Macchiati, 1898 ; Mazzaccara, 1921), whereas the French investigators refer the streaks on the stems to other causes (*Phylloxera*, *Peronospora*, etc.) and consider the bacteria as a secondary factor (Prillieux and Delacroix, 1894, 1895 ; Foëx and Viala, 1894).

In France, where the disease is known as " Gummose bacillaire," several forms have been distinguished, *e.g.* " Dartrose " with small, yellow, scarred streaks on the tendrils and leaf stalks, " Anthracnose ponctuée " with small pustules at the base of the stock, " Rongeot " with reddish discoloured leaves, " Folletage " when the green shoots suddenly die.

The disease was first discovered and described about 1884 in Sicily and Calabria, ten years later it appeared in France and Hungary, and now it has spread throughout Italy.

As preventive measures it is recommended to remove freshly infected shoots in the spring, to cut out old blackened twigs to the base and burn them, and to paint all wounds caused by cutting, etc., with sulphate of iron or some similar preparation.

In other bacterial diseases of the vine the stem may be attacked or the grapes. **" Rogna della Vite "** produces outgrowths on the branches, and these outgrowths, which at first are soft and later hard and woody, contain the *Bacillus Ampelopsoræ* Trer. (Cuboni, 1889). **Vine Tuberculosis** is recognised by the small, grouped tubercles below the bark of the stem, by the dwarfing of the annual shoots and the yellowing of the leaves (Cavara, 1897). **" Roncet "** shows itself first by a weakening of the plant and the splitting of the leaves. If later the stem and root be cut across, dark patches containing colonies of *Bacillus Vitis* Montem. are observed (Pantanelli, 1910, 1911 ; Montemartini, 1913). A sudden drying up of the whole vine, from below upwards, is also ascribed to bacterial action ; such drying up is generally confined to isolated portions of the vineyard and to certain varieties. Vines suffering from this disease are easily broken by the wind. Internally the wood and bark of the stem is blackened and contains numerous colonies of bacteria ; the vine finally dies (Ravaz, 1895).

A bacterial disease of grapes was described in 1891 from Italy. The grapes become brown and finally dry up and fall off (Cugini and Macchiati, 1891). Another disease, which occurred in France in 1895, causes light brown, sunken patches which extend to the seeds, laying them bare and causing them to dry up (Prillieux, 1895).

A bacterial disease of the fig tree has been observed in Southern Italy. The leaves turn yellow and brown patches appear on the young twigs, which later wither and die. In the vessels of the diseased parts a pale yellow, slimy mass containing the *Bacillus Fici* Cav. is found (Cavara, 1905; Petri, 1906).

Bacterial Disease of Mulberry Tree

Bacterium Mori Boy. & Lamb.

This disease gives rise to light-coloured spots, which later become brownish-black, on the twigs and leaves of the mulberry tree (*Morus alba* and *M. nigra*). It attacks young trees in the nursery chiefly, the branches in the head of the tree suffering most. In severe attacks the foliage shrivels and dies, whilst the fruits either drop or only develop incompletely.

The causative organism is *Bacterium Mori* Boy. & Lamb., which is found in large quantities in the affected parts, both in the cells and in the intercellular spaces. The bacteria probably find entrance through cracks or wounds in the bark; at least artificial inoculations have shown this to be possible. The bacteria, after entrance, advance into the inner layers of tissue and spread upwards through the vascular bundles, sending out side branches here and there into the surrounding tissue. Finally, long, dark, sunken streaks appear on the surface of the twigs.

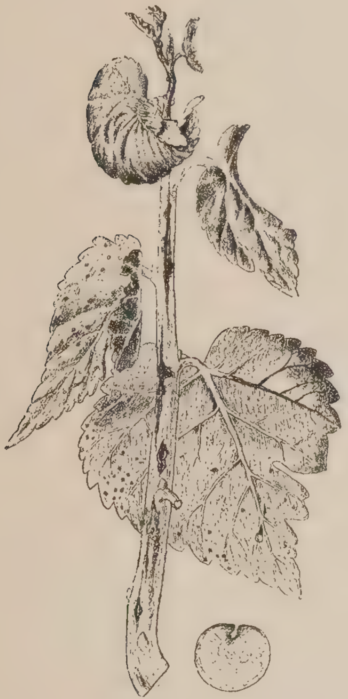

Fig. 27.—Bacterial Disease of Mulberry Tree. (After G. Arnaud and Ch. Secrètin.)

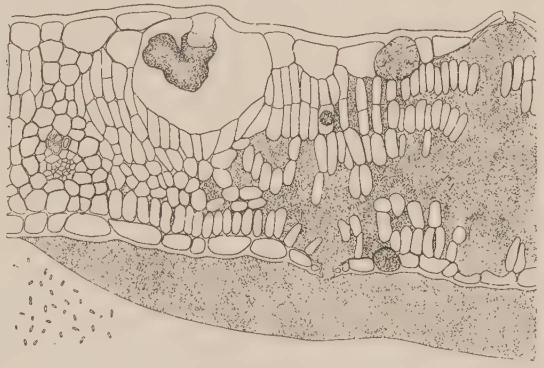

Fig. 28.—Bacterial Disease of Mulberry Tree. Cell tissues containing bacteria. (After G. Arnaud and Ch. Secrètin.)

On the under surface of the leaf there appear large, irregular patches or dark-coloured streaks that follow the larger veins. These patches arise from the centres of infection in the twigs. There is also another form of disease characterized by very small, scattered spots which are surrounded by a light margin. These spots are probably special infections and come from outside. On cutting through these isolated spots they are found to contain bacteria, as does also the viscous material which oozes from the lower surface of the leaf.

This disease was first described in 1890 from Italy, later it was reported from France, Australia (McAlpine, 1898; Osborn and Samuel, 1922), North America, and England.

To combat the disease it is recommended to cut off infected shoots as soon as possible and destroy them.

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Very different in outward characters is "**Necrosis**" of the mulberry tree. This is also a bacterial disease and was observed in Northern Italy (Como) in 1897. Young trees in the nursery suffered on the twigs and stem from flattened canker-like tumours of a brownish-black colour. Black spots, which coalesced, were present on the leaves and the blade of the leaf shrivelled up. Two bacterial forms were isolated from the tumours, one of them identical with *Bacterium Mori* described above, and the other a separate species, *Bacillus Mori-carneus* Cav. (Cavara, 1897).

Bacterial Disease of Strawberry Plants.—In Italy (Turin) a bacterial disease has been found which is characterized by the occurrence, here and there, on the roots of depressed portions bearing white spots. In these spots colonies of bacteria are found. The ground parenchyma and the corky tissue are destroyed (Voglino, 1900).

Another strawberry disease, due to bacteria, is reported from America. Crumpling and darkening of the leaves are characteristic (Stone and R. S. Smith, 1896).

BACTERIAL DISEASES OF OIL-PRODUCING PLANTS

Olive Tree Canker or Olive Knot

Pseudomonas Savastanoi Erw. Sm.

This disease, known in France as "Rogne" and in Italy as "Rogna," appears in the form of irregular nodules or knots of varying size on

2–3-year-old olive trees (*Olea europæa*). The first signs of the disease are apparent in the spring in the shape of transparent patches on the young growing branches. If one of these patches be cut through it is seen that lacunæ of various sizes are present in the bark and cambium tissues, and that they are filled with bacteria and bounded by dead cells. In the tissue between the lacunæ, the individual cells are of abnormal size ; this is due to the action of the bacteria and leads to the enlargement of the lacunæ and the growth of the nodules on the twigs. The nodules are of a corky or woody nature and may attain the size of a walnut, they have their margins furrowed and folded in a variety of ways, the outer side often being deeply sunken. There is also knot formation on the roots. The bacteria can migrate from one centre of infection, by way of the spiral vessels, to other points and there produce further centres of disease.

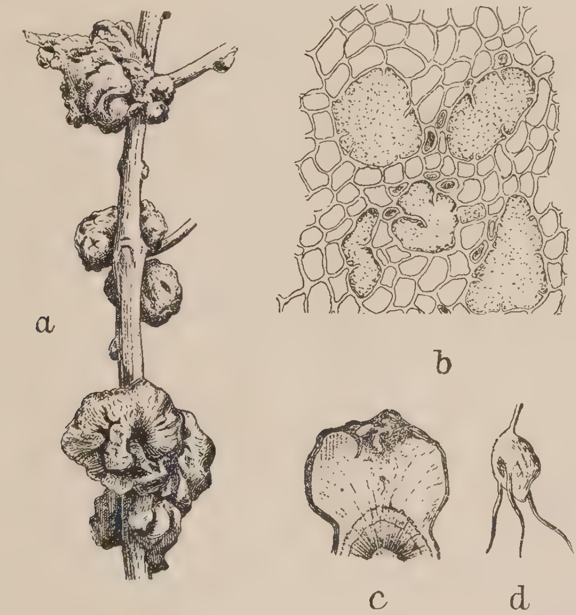

Fig. 29.—Olive Tree Canker.

a, twig with knots ; *b*, young knots ; *c*, root knot ; *d*, cell tissue with bacteria. (*a* and *b* after Prillieux ; *c* and *d* after P. Ferraris.)

Various forms of bacteria and saprophytic fungi are found in the lacunæ and intercellular spaces, the following being the most common : *Bacterium Savastanoi* Erw. Sm. and *Ascobacterium luteum* Bubes, the two always being present together. It has been proved by inoculation experiments that the characteristic knot formation is due to *Bacterium Savastanoi*.

It is noteworthy that the bacteria responsible for the disease live symbiotically with the olive fly (*Dacus Oleæ*), which is a source of great danger to the olive crop in all the Mediterranean countries. The larvæ of this fly always harbour the bacteria in the intestine, and they remain there until the pupal stage is reached. In the absence of the bacteria the larvæ can hardly develop to the imago stage (Petri, 1909). It is, therefore, easily understood that the fly contributes to a considerable degree to the distribution of the knot disease.

By artificial inoculation experiments with material taken from the nodules, it has been possible to produce similar growths on *Fraxinus velutina*, *F. floribunda*, *Adelia acuminata*, and *Jasminum primulinum*, whilst species of *Carissa*, *Chrysanthemum*, *Coprosnea*, *Eleagnus*, *Nerium*, *Osmanthus*, *Prunus*, *Syringa*, *Thevelia*, *Vinca*, etc., are immune. It has been suggested that tubercular growths observed on *Fraxinus excelsior* at Vogelsberg (Hesse-Darmstadt) are due to the same organism—*Bacterium Savastanoi* (Noack, 1893).

The severe losses which this disease causes to the olive growers were not discussed until the end of the last century, when reports on the subject were published in France, Italy, California, and other countries.

Preventive Measures :

(1) Wounds and injuries arising from the taking of cuttings from the young olive trees should as far as possible be guarded against, as they serve as points of entry for the bacteria. (2) Healthy trees must not be pruned with knives, etc.. which have previously been used on infected trees, until the instruments have been properly disinfected. (3) Resistant varieties should be selected for new plantations.

Similar tubercles have been observed on *Nerium Oleander* and ascribed also to *Bacterium Savastanoi*. In one case the tubercles appeared on the twigs, on some leaves, and on the flower stalks, particularly, however, on the flowers after fertilization (Peglion, 1905). In another case, warts of different sizes and with cracked margins were formed on the woody twigs and green shoots (Tonelli, 1913). More recently a similar disease has been reported from the South of France (Arnaud, M. and G., 1923).

A bacterial disease, closely related to the knot disease of the olive tree, if not identical with it, was described from the South of France a long time ago. The disease produces similar knots on the Aleppo pine (*Pinus halapensis*), and the organism has been named *Bacterium Pini* Vuill. (Vuillemin, 1888 ; Prillieux, 1890).

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Bacterial Disease of Sesame.—A bacterial disease has been observed on *Sesamum orientale* growing in seed beds in Bulgaria. Dark brown to black spots appear on the leaves and afterwards on the stems. A thick, slimy fluid oozes from the diseased parts of the stem and dries to a greyish-black mass. The infected stems finally fall over. The disease is caused by two organisms in symbiosis—*Bacillus Sesami* Malk., which has yellow colonies of bacteria, and *Pseudomonas Sesami* Malk. in grey colonies. Each of these forms can produce the disease independently of the other. Steeping the seed in 1 per cent. formaldehyde solution has given good results (Malkoff, 1904, 1906).

A bacterial disease on *Ricinus communis* has been reported in the United States (Florida, Carolina, Mississippi) since 1918. In slight attacks, individual leaves fall from their stalks, in severe cases the whole plant, with any inflorescences it may be carrying, collapses. The disease was worst on newly cultivated soil and is said to be caused by *Bacterium Solanacearum* Erw. Sm. (Erwin Smith and Godfrey, 1921).

BACTERIAL DISEASES OF ORNAMENTAL PLANTS

Bacterial Disease of Lilac

Pseudomonas Syringæ Beijer.

This disease appears on the stem, branches, and leaves of young lilac trees. Long streaks of brown to black spots are produced at the internodes of the branches or twigs, and in a warm, damp atmosphere the branch is girdled by the extension of the streaks and bends over. The nature of the attack on the leaf varies according to whether the infection has come from the axis, through the vascular bundles, or whether the leaf has been infected by contact with other diseased parts. In the first case brownish-black stripes are produced along the larger leaf veins, whilst in the second case spots appear irregularly over the leaf blade and finally the tissue at these spots dries, leaving holes. On cutting across one of the young centres of disease found on the stem, large turbid masses of the organism—*Pseudomonas Syringæ*—are seen to be present in the intercellular spaces or in cavities which have arisen through general dissolution of cell tissue.

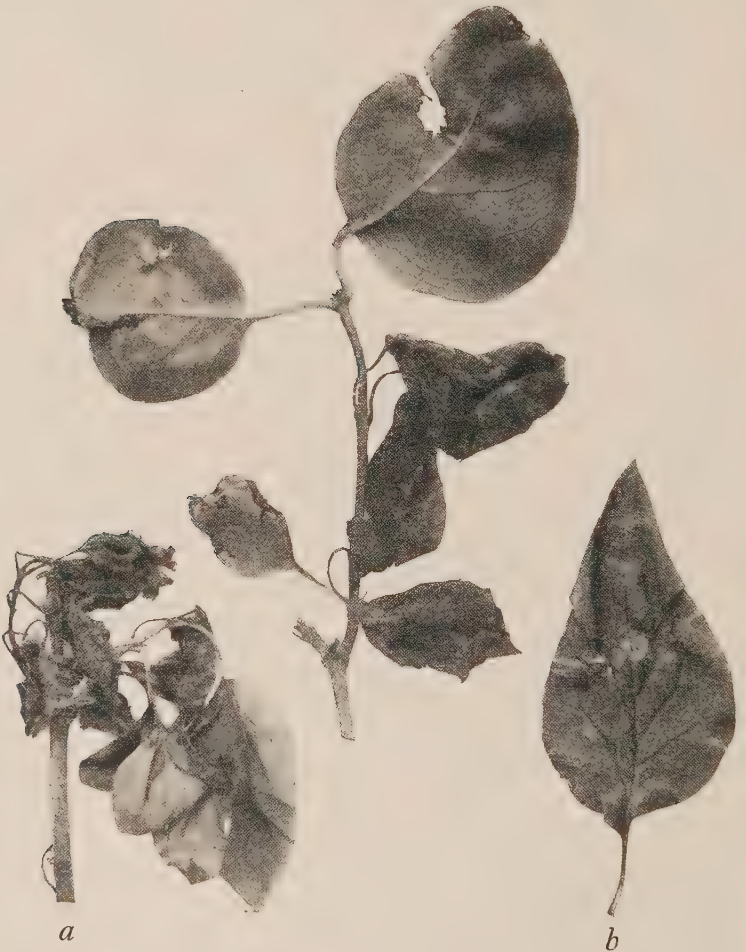

Fig. 30.—Bacterial Disease of Lilac.
a, natural infection ; *b*, artificial infection. (Orig.)

When lilac is grown out of doors, the disease appears in May to June, and under glass in February to March. A warm, moist atmosphere favours the growth of the bacteria and the development of the disease. The diseased parts of the plant adhere together and the flowers only develop sparingly. If, however, dry conditions prevail during the first stages of the disease the plant may recover.

It has been observed in Holland that the single-flowered lilacs are the ones that are mainly attacked, especially strains of *Syringa vulgaris*. Artificial inoculation experiments have shown that the disease can be transmitted to *Populus nigra*, apple, pear, *Prunus Mahaleb*, *Polygonum*, *Fagopyrum*, and *Atriplex hortensis*, whilst *Sorbus Aucuparia*, *Quercus Cerris*, *Cytisus Adami*, *Deutzia scabra*, species of *Spiræa*, etc., have proved immune.

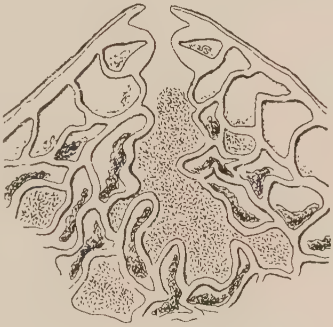

FIG. 31.—Bacterial Disease of Lilac Leaf Tissues with Bacteria. (After H. Klebahn.)

The disease can be transferred from place to place in infected plants and cuttings. Thus, in Sweden it was found that special sorts of lilac imported from Holland and grown under glass were attacked by the disease each spring, whilst home-raised sorts were quite free. It was found possible in the end to keep down the disease by lowering the temperature and moisture conditions in the glasshouse.

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Crown Gall of Chrysanthemum frutescens

Bacterium tumefaciens Erw. Sm.

This disease, which appears on certain sorts of *Chrysanthemum frutescens*—such as "Mm. Sander"—gives rise to a collection of tumour-like growths round the crown of the root. These root galls may be as large as a plum or a peach, whilst on the aerial parts of the plant elongated excrescences may be found.

The galls are produced by *Bacterium tumefaciens* Erw. Sm. & Towns., which causes excessive cell division in the tissues that are attacked. The same bacterium, or biologic forms of it, produces similar galls on the roots of other plants such as the mangel (p. 20), and species of the genera *Rosa*, *Medicago*, *Salix*, *Pyrus*, etc. The bacterium has been isolated from thirty four different host plants belonging to fourteen families.

The disease was first observed in North America at the beginning of the present century; later it was reported from various European countries—in 1912 from Italy and Sweden; 1913 from Germany and France; 1921 from England.

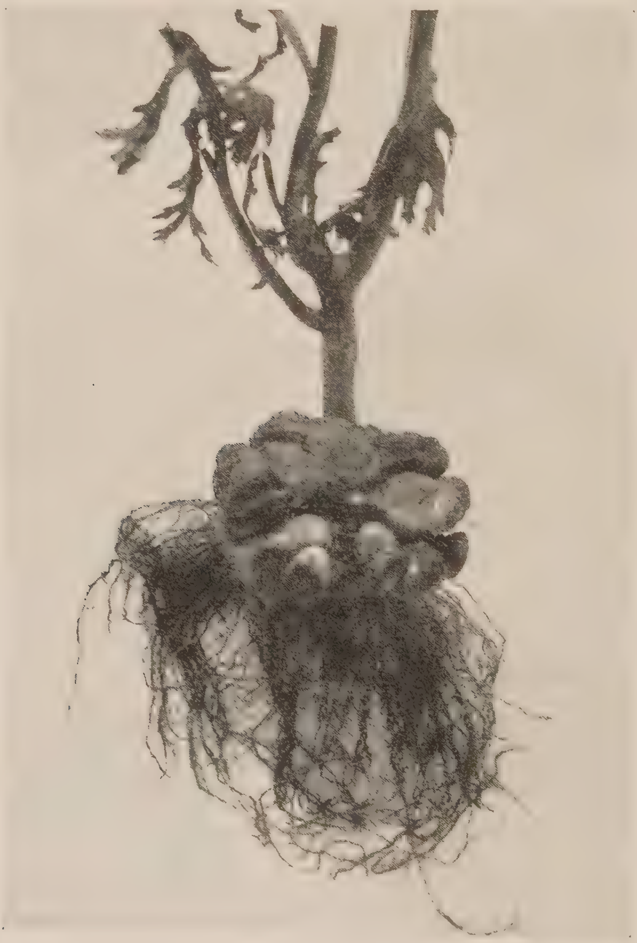

FIG. 32.—Crown Galls on *Chrysanthemum frutescens*. (Orig.)

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Bacterial Disease of Stocks.—The fungus, *Bacterium Matthiolæ* Briosi & Pavar., is found on *Matthiola annua* and *Cheiranthus Cheiri* and causes the leaves on the lower part of the stem to fall, whilst those on the upper part become greenish-yellow and flaccid. On examination, the veins of the leaves are seen to be of a dark, almost black, colour, due to the destruction of the vascular bundles; these dark strands are also seen when the infected petiole or stem is cut across. The inflorescences wither, and cultivation of the flowers becomes unprofitable. Large numbers of bacteria are found in clumps in the infected tissues.

In 1900 the disease was observed on summer stocks in Holland, in 1904–6 on winter stocks in Bavaria, and in 1910–11 it caused considerable damage to crops of *Matthiola* and *Cheiranthus* in Liguria (van Hall, 1900; v. Faber, 1907; Briosi and Pavarino, 1912).

Bacterial Diseases of Cyclamen.—At the end of the last century a disease was observed in France on *Cyclamen persicum*, the leaves and flowers of which withered and finally died (Prillieux and Delacroix, 1894). Recently a similar disease, "Cyclamen Wilt," has been observed in England, several cases having occurred since 1922. The first signs of the disease are a wilting and incurving of the leaves, the oldest ones being attacked first. The roots are also affected, becoming brown and rotten, whilst the rhizome is changed into a spongy, black mass (Butcher, 1925). A similar disease was noticed about the same time in Liguria (Montemartini, 1924).

Ivy Canker attacks the stems and leaves of ivy sets (*Hedera Helix*) and finally causes them to die; masses of bacteria are found in the canker lesions. The disease was found in the neighbourhood of Berlin (Lindau, 1894). It has been possible to inoculate *Hedera* and *Tropæolum* with bacteria, in which the form *Microspira carcinopœa* Rég. was present, taken from a canker on a young oak tree, and produce in them secondary lesions (Régamey, 1914).

Bacterial Diseases of the Arææ.—Both in the glasshouse and out of doors a distinctive disease due to bacteria sometimes occurs very destructively on *Calla æthiopica*. The tuber becomes brown, soft, and watery, the tips of the leaves turn brown and dry, and soon the whole surface of the leaf is marked with brown spots of a considerable size. Often the leaves bend over sharply and the flower stalk collapses in the same way. Bacteria are found in enormous numbers between the remaining parts of the cells in the infected tissues. When the weather is moist and warm the tuber rots within three or four days. The organism which causes the disease is *Bacillus Aroideæ* Towns.

The disease was first observed in the United States in 1893 (Halsted) and examined and described in 1904 (Townsend). A similar disease on *Calla Childsiana* has been found in Holland (Ritzema Bos, 1914), and on *Arum sp.* in England (Bewley, 1922, 1925).

A bacterial disease has been found on *Calocasia esculenta* in America; the tuber and leaves are changed into a foul-smelling, slimy mass. *Bacillus carotovorus* Jon. is given as the cause of the disease (Harter, 1916).

White Rot or Yellow Disease of Hyacinth

Pseudomonas Hyacinthi (Wak.) Erw. Sm.

This disease, which is endemic in Holland, manifests itself at the time when the leaves of the plants are drying off or even earlier. Apparently healthy plants, in which the flower buds seem to be developing normally, suddenly come to a standstill. The flower shoot ceases to extend and the flowers themselves develop irregularly. Some of the leaves soon turn yellow at the tip and the yellow colour gradually passes, often in streaks, to the region of the vascular bundles moving downwards to the base of the leaf, whilst the tips begin to dry off. Generally other leaves of the same plant are infected and the infection spreads, in about a fortnight, to the leaves of the daughter bulb. At the time when the bulbs first begin to grow it is difficult to detect the disease owing to the leaves being tightly closed. When the disease has reached an advanced stage the centre bulb scales are rotten at the base, and it is in these rotten patches, which have an unpleasant smell and are found in the intercellular spaces and cavities (due to the breaking down of cell tissues), that the bacteria are found. The bacterium causing the disease is *Pseudomonas Hyacinthi*, which is rod-shaped with rounded ends. It has one polar flagellum by means of which it moves about in young cultures.

Sometimes in artificial cultures the bacteria form segmented chains of 6–12 bacteria or unsegmented threads of greater length.

In the bacterial slime, at least in the later stages of the disease, mycelial hyphæ are found, and these are considered to belong to a fungus of the genus *Hypomyces* (*H. Hyacinthi* Lor.). It is assumed that these hyphæ serve to transmit the bacteria from a diseased plant to a healthy one in the field.

Manifestations of disease similar to those just described are met with in bulbs which have been taken from their growing place and laid in the ground to ripen off, especially if the rainfall is considerable and the soil warm. Such bulbs, if badly diseased, look as though they had been boiled and are changed to a gelatinous, stinking mass. The same symptoms also appear in bulbs lying on racks in the storeroom, and the yellow slime may cover the boards and glue down the bulbs. Such bulbs can carry the disease to other places, as many recorded instances prove (Weber, 1924).

It has been long known that certain sorts of bulbs are more or less resistant to the disease. In 1885 a list based on the experience of the Dutch hyacinth growers was prepared, in which 8 varieties were given as very resistant and 13 as very susceptible. In 1906 three of the chief Dutch growers unanimously agreed that of the well-known varieties only 6 (Robert Steiger, Grandeur à Merveille, Grand Maître, La Peyrouse, King of the Blues, and Marie) had proved to be very resistant, and that 7 (La Grandesse, La Neige, Zar Peter, Grand Lilas, Lord Derby, Hermann,

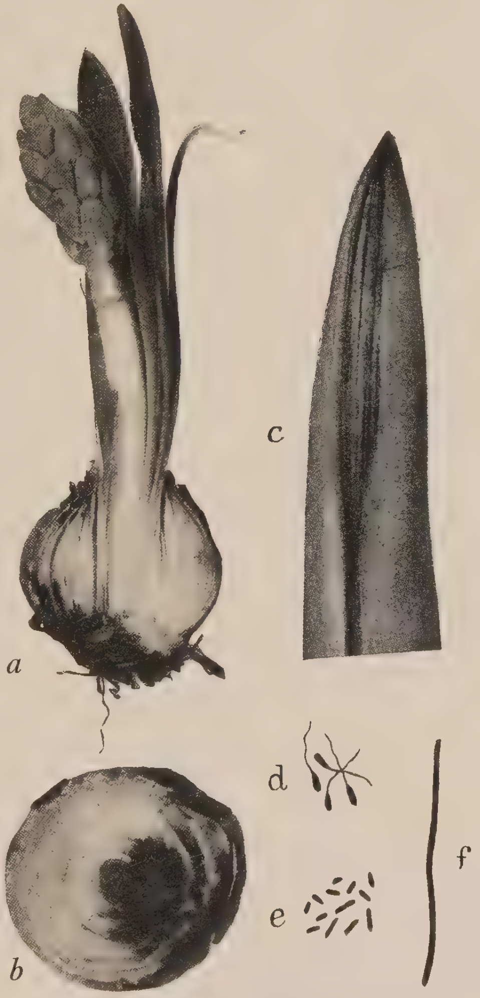

Fig. 33.—White Rot of Hyacinth.

a, longitudinal section of plant; *b*, cross-section of bulb; *c*, infected leaf; *d-f*, forms of bacteria. (*a* and *b*, Orig.; *c*, *f*, after Erwin Smith.)

and La Tour d'Auvergne) were very susceptible, whilst in the case of 154 varieties their experience varied.

The disease has been transmitted by artificial inoculations to the leaves of *Hyacinthus albulus*, *Allium Cepa*, *Amaryllis atamasco*.

The following preventive measures, based upon Dutch experience, are recommended: (1) By the prompt removal of the first diseased scales the spread of the disease in the bulbs may be checked. (2) The implements (spades, knives, etc.) used in infected crops must be carefully sterilised before being taken on to a clean field. (3) Badly diseased plants should be dug up and destroyed as soon as noticed. (4) In the selection of bulbs, regard should be had to those which are resistant to this disease.

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A Bacterial Disease of Iris has been reported on *Iris florentina* and *I. germanica* in Holland. The wing shoots, when only a few centimetres above the ground, become brown and withered at the tips. This drying up gradually spreads to the lower part of the shoot, and the plant finally dies. The base of the leaf lying in the soil, and the part of the rhizome belonging to it, rot to a pulpy yellowish-brown mass.

Sometimes in the course of the summer the disease invades the whole of the rhizome, so that there remains only a pulpy mass of a clear white colour and a mouldy smell.

Several forms of bacteria can cause the disease. Thus in one investigation in 1901 *Pseudomonas Iridis* v. Hall was found, and later in the year on the same field *Bacillus omnivorus* v. Hall. In 1902 fresh material from the same field yielded a feebly fluorescent species, *Pseudomonas fluorescens exitiosus* v. Hall, together with *Bacillus omnivorus*, whilst *Pseudomonas Iridis* was entirely absent. Exhaustive inoculation experiments have shown that all three species—and each alone—can produce the disease, but that *Bacillus omnivorus* is the chief agent. The main action of the bacteria is to destroy the individual cells of the tissues and to consume the contents which diffuses from them.

The young organs of the host plant are always more susceptible than at a later stage, and the conditions under which the plant is living have a considerable influence upon susceptibility. There is reason to think that in cases of this kind the bacteria were originally saprophytic, but as a result of cultural conditions have become " facultative parasites " (v. Hall, 1903).

In Austria, similar forms of bacteria have been met with on *Iris pallida*, where the rhizome was attacked (Heinricher, 1902), and in Italy (Florence), where the disease manifested itself by a yellowing of the edges of the leaves, afterwards on the leaves themselves, then on the thickened base of the flower stalk, and finally the rhizome became infected (Cavara, 1911).

Bacterial Diseases of the Orchidaceæ.—A number of tropical members of the family Orchidaceæ growing in glasshouses have recently been observed in Italy to suffer from bacterial diseases. In the case of *Cattleya Werneri* and *C. Harrisoniæ* brown spots and rust-coloured pustules occurred in which colonies of *Bacterium Cattleyæ* Pav. were present. The leaves of *Odontoglossum citriosmum* showed characteristic spots containing *Bacterium Pollacii* Pav. Transparent areas on the leaves of *Oncidium Kramerianum*, which by transmitted light had a rust colour, were caused by *Bacterium Krameriani* Pav. In diseased specimens of *Oncidium ornithorynchum* and *Cattleya crispa* the *Bacterium farnetianus* was present. On the leaves of *Vanilla planifolia* pitch-black spots occurred which led to the puncturing and tearing of the leaf tissue ; the organism was *Bacterium Briosianum* Pav. (Paverino, 1911).

Bacterial Disease of the Poplar. -A canker-like disease was observed in Italy in 1906 on *Populus alba*, *P. nigra*, and *P. tremula*. The disease attacked the stem and branches of one- and two-year-old trees, producing round, smooth outgrowths about the size of a pea. These growths gradually became brown and furrowed on the surface, and by lateral development finally girdled the branch. The disease is caused by *Micrococcus Populi* Brizi (Brizi, 1907). Recently the same disease has been observed in France and Belgium on *Populus regenerata* (Hiley, 1923).

CHAPTER II

MYXOMYCETES—SLIME FUNGI

The slime fungi, which form a completely separated group in the plant kingdom, show a distinct relation to the protozoa of the animal kingdom. The fungi in this group are not composed of true cells or hyphæ, but consist of a mass of naked protoplasm—a plasmodium—which has no definite shape. The plasmodium, which is capable of movement over its substratum, is sometimes colourless but may be yellow, red, bluish-black, or violet in colour. When the vegetative stage is complete the plasmodium is changed into a large number of spherical spores, which are either contained in sporangia (endospores) or are formed on the surface (exospores). When a spore germinates it produces a swarm spore (myxamœba) which is able to change its form continually, and is capable of a creeping movement. The plasmodium obtains nourishment from the substratum, which may be either living or dead, upon which it lies. By the fusion of a number of plasmodia a considerable mass of protoplasm may arise.

Most of the slime fungi live on an inert substratum, *e.g.* rotting stumps, withered leaves, dead wood, and may spread from these to adjacent plants, where they cause injury by depriving the plant of light and air. The saprophytic forms usually possess a spore receptacle, or capillitium, with a wall composed of dried mucous within which is a network of closely-woven threads.

A small number of slime fungi are parasitic upon living plants, and in such cases the spores are not surrounded by a common membrane but lie within the cells of the host plant.

Club Root of Cabbage, etc.

Plasmodiophora Brassicæ Wor.

This disease shows itself in summer in cabbage or turnip crops by the stunted, unhealthy appearance of some of the plants. If the root of one of these plants is examined a number of swellings of varied shape and size are seen. These swellings give a distorted appearance to the root, and they remain white and hard throughout the summer, but in autumn or winter the inside decays, producing a dark-coloured, foul-smelling mass.

If during the summer one of the "clubs" or swellings be cut across and the inside examined under the microscope, it will be seen that certain cells are larger than the surrounding ones and show a colourless, granular contents. With a sufficiently high magnification this granular material can be seen to be composed of intra-cellular plasmodia, and it is they which are the cause of the disease. The plasmodium divides up into numerous spherical spores which are liberated into the soil when the infected root decays. The spores remain unchanged throughout the winter and germinate in the spring, the contents of the spore escaping as a naked mass of protoplasm, the myxamœba or swarm spore, which is capable of movement and of change of form.

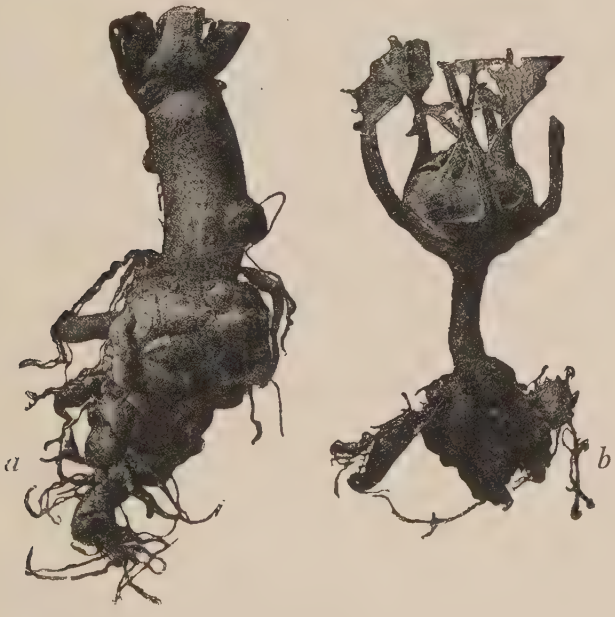

FIG. 34.—Club Root.
a, turnip; *b*, kohl-rabi. (Orig.)

When such a myxamœba comes in contact with the root of one of the plants susceptible to the disease it bores its way into the epidermal tissue. The penetration of the slime fungus as well as its passage from cell to cell within the root appears to be possible without there being any opening in the cell wall; at least, no hole can be detected after the fungus has passed through (Kunkel, 1918). Many of the spores present in the soil retain their viability for a number of years, and it has been proved that an infected soil may remain so for three and possibly five years.

The disease is carried from an infected field to a clean one on implements, hoofs of animals, or men's shoes; also in manure from animals that have been fed on uncooked infected roots. The manure is especially dangerous when uneaten portions of roots are taken from the feeding troughs and thrown on to the manure heap.

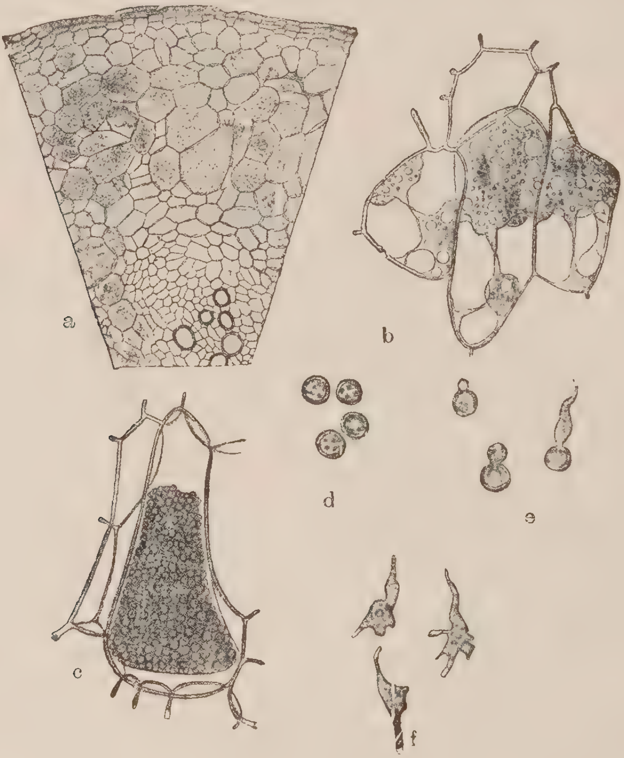

Fig. 35.—Club Root.

a, section of diseased root; *b*, cells from swelling on root showing plasmodia; *c*, cells containing spores; *d*, spores; *e*, germinating spores; *f*, swarm spores. (After M. Woronin.)

Outbreaks of disease vary from year to year and from place to place. A damp situation favours the development of the disease, whilst soils rich in lime check it. In some cases the infection seems to be seed-borne, as was observed in one case in Sweden in 1909, where turnips raised from purchased seed were diseased whilst those of the variety usually grown were healthy, although quite near and in much larger numbers.

The disease not only attacks different varieties of *Brassica oleracea*—cabbage, cauliflower, brussels sprouts, kohl-rabi, etc.—but other members of the cabbage family, such as swedes (*Br. Napus*) and turnips (*Br. Rapa*). Further, the disease has been found on other *Cruciferæ*, both cultivated and wild ; in the former group are *Camelina sativa*, *Cheiranthus Cheiri*, *Hesperis matronalis*, *Matthiola incana*, *Raphanus rhaphanistrum*, *Sinapis alba*, etc., whilst among the wild ones are *Capsella bursa-pastoris*, *Cardamine pratensis*, *Erysimum cheiranthoides*, *Sinapis arvensis*, *Sisymbrium officinale*. According to Danish experience, turnips on sandy soils suffer most and swedes on loamy soils least ; also that amongst turnips the round sorts are less affected than the long-shaped ones.

The history of this disease, which is also known as "Finger and Toe," and "Anbury," can be traced back to 1736, when it was first mentioned in England. It was not, however, until 1820 that it was described as a pest of considerable importance, and since then it has been reported from many countries as causing very serious damage.

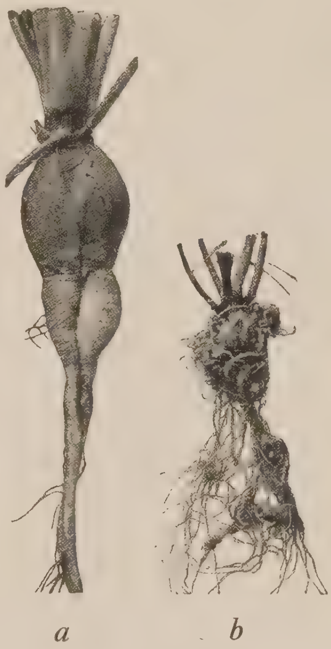

Fig. 36.—Club Root.

a, *Raphanus sativus* ; *b*, *Capsella bursa pastoris*. (Orig.)

Preventive and Control Measures.

(1) Soil on which crops of the cabbage family are to be grown should be well drained. (2) Fields in which "clubbed" plants have appeared should not be used for similar crops for 4–6 years, but instead potatoes, peas, clover, cereals, etc., should be grown. It is also important to eliminate such cruciferous weeds as *Sinapis*, *Thlaspi*, *Capsella*, *Erysimum*, etc., as the fungus, in case of need, can live upon them. (3) If an infected field must be used for a susceptible crop, a heavy dressing of ground lime—say 1 ton per acre—should be applied some time ($\frac{1}{2}$–$1\frac{1}{2}$ years) before it is intended to sow the crop in question. (4) To prevent the transfer of infected soil to clean land, all implements and tools, as well as the hoofs of animals and shoes of labourers, should be thoroughly cleaned. (5) The soil used in seed beds must be above suspicion, and the boxes, frames, etc., in which the seedlings are raised must be carefully cleaned, or better, washed with a disinfectant solution such as a

tar-distillate fruit wash (1 part in 100 parts of water). The seedlings should also be examined for swellings. (6) Varieties which have shown resistance to the disease should be selected and grown. (7) If slightly infected roots are used for feeding, the uneaten portions should on no account be thrown on to the manure heap. If possible diseased roots should be boiled before being fed. Any suspicious refuse from the garden or house should not find its way to the manure heap. (8) Diseased roots that are not to be fed should be thrown into a pond or lake, where they can do no harm ; on no account should they be put on the manure heap. (9) As manure, nitrate of soda is preferable to sulphate of ammonia and basic slag to superphosphate.

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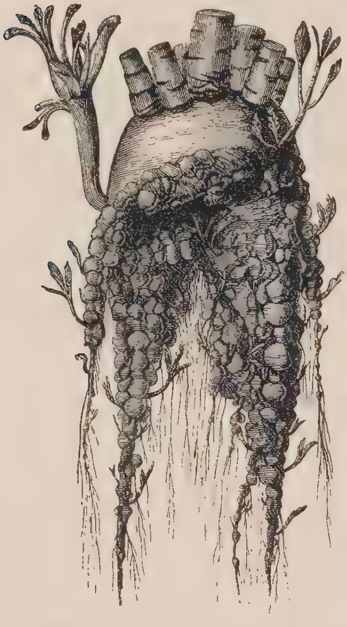

Fig. 37.—Root of a cross between *Brassica campestris* and *B. Napus*. (After Samsoe-Lund and Kiaerskou.)

Misshapen roots of non-parasitic origin are sometimes found in a crop and must not be confused with "clubbed" roots. Such misshapen roots often exhibit numerous small nodules on the lower parts ; these nodules can free themselves and develop into another plant which produces seed which, in its turn, gives plants with similar nodules on the roots. They have been found, growing naturally in the open, in Germany (Caspary, 1873), and in Denmark they have been produced by the artificial crossing of *Brassica campestris* and *B. Napus* (Samsoe-Lund and Kiaerskou, 1879).

"Club Root" of Vine.—This disease, known in France as "Brunissure," gives rise to light brown angular patches—which may be of considerable size—on the upper surface of the leaf between the veins. The patches, which have sharply-defined margins, gradually increase in size until the whole of the leaf surface, except the edges and the veins, is covered. The under surface of the leaf remains normal or may be slightly reddened. Ultimately the leaf becomes of a rusty-red colour, decays, and falls. The disease is caused apparently by *Plasmodiophora Vitis* Viala and Sauv., which lives chiefly in the palisade cells and later also in the spongy parenchyma, but only rarely in the epidermis. The plasmodium appears to pass through pores in the cell walls.

The disease has been known since 1882 in vineyards in the south of France. In 1892 it was observed in Italy and in 1893 in England, where it caused swellings or "clubs" on the root similar to those produced on the cabbage by *Plasmodrophora Brassicæ* (Viala and Sauvageau, 1892; Viala, 1893; Cooke, 1893; Cavara, 1894; Moritz and Busse, 1894; Debray, 1898; Viala and Sauvageau, 1898).

Closely related to the above is a disease which appeared in South California in 1882 and has been the cause of the destruction of large areas of vines. The disease, which also attacks the wild vine (*Vitis californica*), appears in early spring on the tips of the shoots and spreads downward to the root. The shoots develop with short internodes and are much branched; in autumn the wood vessels in the stem show brown zones; the mottled leaves wither prematurely and fall. The fungus has been named *Plasmodiophora californica* Viala and Sauv. (Pierce, 1890; Viala and Sauvageau, 1892; Viala, 1893).

Corky or Powdery Scab of Potato

Spongospora subterranea (Wall.) Johns.

This disease attacks small potato tubers, even when they are no larger than a hazel nut, and gives rise to small, smooth, wart-like swellings which are covered by the skin of the potato. These warts are at first few in number, and if the soil is moderately dry a layer of cork is formed

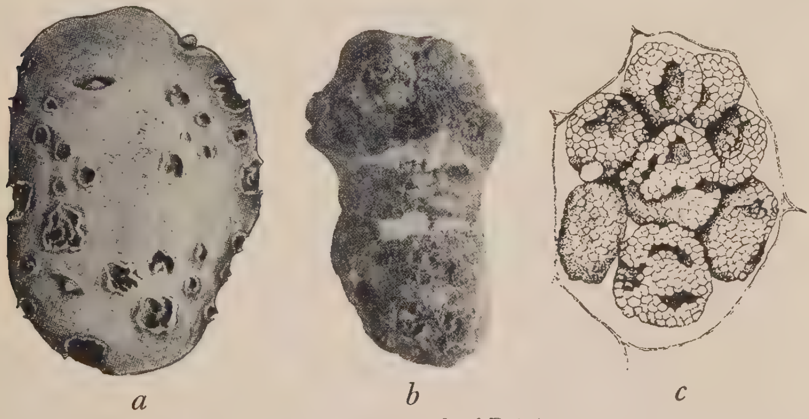

FIG. 38.—Corky Scab of Potato.

a, slightly diseased potato; *b*, misshapen potato, severely attacked; *c*, cells containing spore-balls inside the skin of potato. (*a*, after T. Johnson; *b*, after H. T. Güssow; *c*, after T. B. G. Osborn.)

below them and the disease is stopped. On the other hand, in a very damp situation there is no cork formation and the warts increase in size with the growth of the potato. They may attain a circumference of 3–4 cm. and penetrate into the flesh to a depth of 2 cm. The warts gradually give place to depressed scabs, several of which may coalesce. Tubers that are severely attacked often become irregular in shape. At the bottom of the scab cavity a fine, brown powder, which is composed of resting spores (spore-balls), is found for a time, and this condition has led to the disease being called "Powdery Scab." Each spore-ball is composed of a large number of angular spores arranged closely in rows with spaces between them.

The further development of the fungus is as follows: the individual spores from the spore-balls separate from one another when ripe and germinate, producing a myxomœba which may lead a separate existence, but generally unites with others to form a plasmodium. After becoming free from the surrounding cell membrane the plasmodium, together with those from other cells, develops as an intercellular plasmodium directly below the skin of the potato, until finally it penetrates a previously uninfected cell by means of delicate threads (pseudopodia). From this point onward the fungus leads an intracellular life. The protoplasm of the fungus is so intimately associated with that of the host cell that the two cannot be distinguished, and in this symbiotic condition they pass from cell to cell through the wall.* The entire contents of the host cells (starch grains, nuclei, etc.) are gradually used up by the

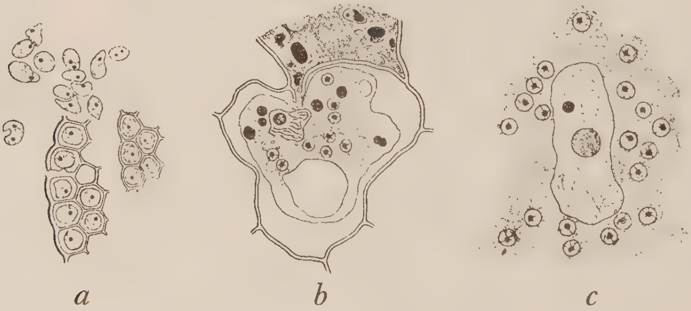

Fig. 39.—Fungus of Corky Scab.

a, group of resting spores, partly ruptured and forming myxamœbæ; *b*, cells of potato infected by plasmodium; *c*, plasmodium surrounding cell nucleus, with numerous nucleoli. (After L. O. Kunkel.)

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* This symbiosis between two plasm bodies has a close similarity to a condition, to which the name of "Mycoplasm" has been given, observed in certain *Peronosporaceæ* (see p. 74) and *Uredinaceæ* (see p. 147).

plasmodium, while at the same time numerous nuclear bodies (nucleoli) are formed in the plasmodium, and these can be regarded as the foci of fresh generations of spore-balls.

The predisposing conditions for an outbreak of disease are (*a*) alkaline reaction of the soil ; (*b*) cool, moist, misty weather following continued rain during the growing season, especially if the soil is not well drained ; (*c*) a relatively high soil temperature during growth.

The disease has been traced back to 1842, when it occurred in Germany (Pethybridge, 1913). In later times—from 1887 onward—it has been found in Norway, Ireland, Denmark, the United States, etc.

Preventive and Control Measures :

(1) Diseased potatoes must not be used for seed. (2) If slightly affected tubers, or those grown on infected soil, have to be used, they should be disinfected by means of corrosive sublimate solution (1 : 1000 or 1 : 1500) or in formalin (diluted about thirty times with water) for 1½ hours. (3) Those varieties of potatoes that have shown themselves locally to be resistant to the disease should be chosen. (4) A long interval—three, or better six to seven, years—should elapse before potatoes are grown again on infected soil. (5) As the spores of the fungus remain viable after passing through the intestine of the pig, the dung from animals that have been fed with uncooked, scabby potatoes should not be used on arable land.

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The following slime fungi, which are essentially found on grass meadows and are indirectly injurious, deserve mention : *Physarum cinereum* Pers., found in Sweden (near Jönköping) in 1905. The stems of the grass were covered with a slimy material and became first greyish-white in appearance and finally black. Owing to the attack of the fungus the development of the stems was checked (Wulff, 1906). In the same district another slime fungus, *Spumaria alba* Bull., appeared in 1906, not only on grasses but also on other species of plants, such as *Lamium album* and *Ulmus montana*, producing on them white or greyish-black masses of slime (Wulff, 1908).

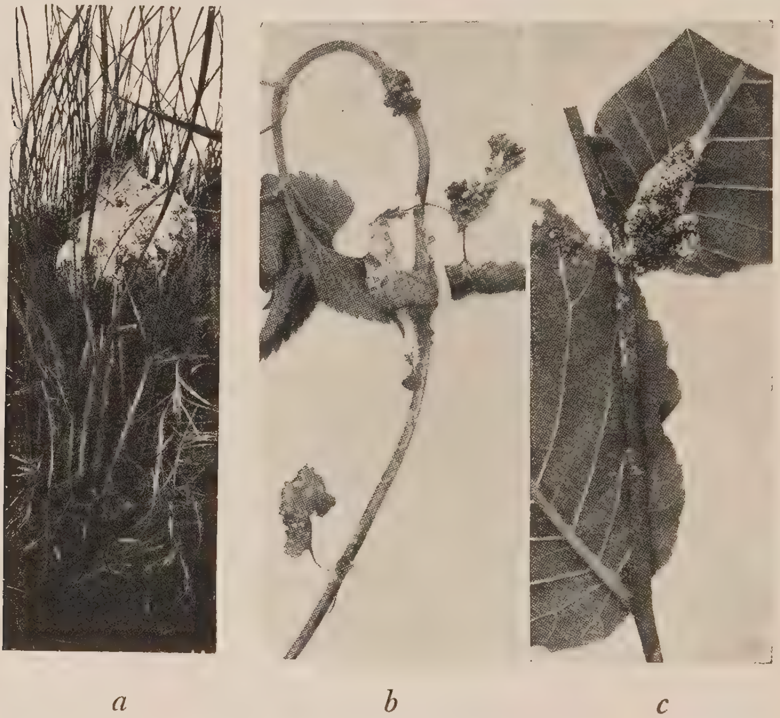

Fig. 40.—*Spumaria alba.*
a, on grass ; *b*, on *Lamium album* ; *c*, on *Ulmus montana*. (Orig.)

CHAPTER III

EUMYCETES—TRUE FUNGI

These fungi possess a mycelium composed of hyphæ which collectively form the vegetative part of the fungus—the thallus—from which the reproductive structures—the spores—are developed.

OOMYCETES

Non-septate mycelium. Sexual reproduction. Differentiation between male and female sexual cells.

Chytridiaceæ

The members of this order are the lowest of the fungi possessing a mycelium. The mycelium, at least at first, is non-septate, and in some cases septa are never found. Each new fungous individual arises from a spore which attaches itself to the outside of one of the cells of the host plant ; from this point it penetrates into the interior of the cell and there completes its development. Most of the fungi that belong here are aquatic and exist as parasites upon algæ ; only a few are parasitic upon the higher plants.

Wart Disease of the Potato

Synchytrium endobioticum (Schilb.) Perc.

This disease, also known as " Black Scab," can be recognised by the warty growths which develop upon the newly formed tubers about the time when the plant is in flower, and they may also appear on the stolons and roots. As a rule the warts, when fully developed, are as big as a walnut or even bigger, and in severe attacks a large, irregular tumour-like mass is produced. The warts are at first white, but later they become dark brown or black. If the diseased tubers are stored in a damp place they quickly begin to rot, and ultimately pass into a putrid mass.

The disease is caused by the fungus *Synchytrium endobioticum* (Schilb.)

Perc. (the name first given to it was *Chrysophlyctis endobiotica* Schilb.). The fungus lives within the cells of the potato, principally in the tissues just below the skin. In young tissue the fungus appears as an indistinguishable symbiotic plasmodium which has been formed by the amalgamation of the protoplasm of the fungus with that of the host cell. It is only at a later stage that the fungus appears as a separate structure. In every diseased cell a plasmodium is found, and this gradually changes into a number of golden-brown spore receptacles (sporangia) containing a very large number of zoospores, that serve for the spread of the disease during the growing period of the plant, or else into a single thick-walled resting spore which serves to keep the fungus alive until the following year. The resting spores either pass into the

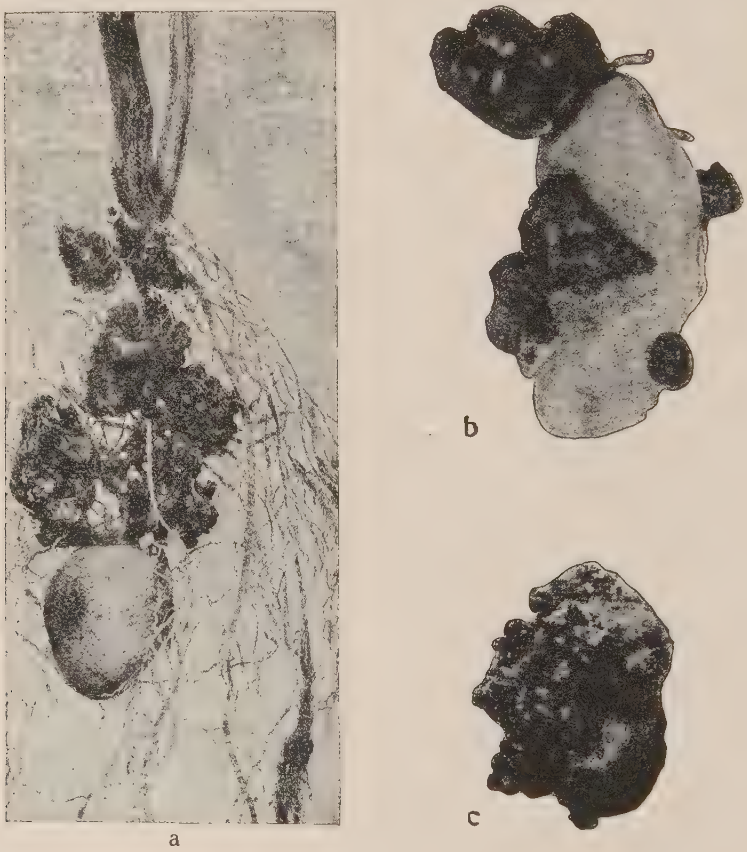

Fig. 41.—Wart Disease of Potato.
a, underground portion of diseased potato; *b* and *c*, diseased tubers.
(*a* and *c*, after O. Appel; *b*, after T. Johnson.)

soil when the potato decays, or they are carried on slightly infected tubers (should such be used as seed). Infected soil can impart the disease to potatoes for at least two years, perhaps even as long as six to seven years. Young tubers are infected in the "eyes," and the irritation which is thus produced causes abnormal development of cell tissue and the formation of cancerous outgrowths.

Artificial inoculation has resulted in the transmission of the disease to *Solanum nigrum* and *S. Dulcamara* (Cotton, 1916) and to tomatoes (Kunkel and Orton, 1920), whilst numerous species of the genera *Solanum*, *Capsicum*, *Datura*, *Physalis*, *Nicotiana*, etc., have shown themselves to be immune (Weiss, 1923).

The disease was first detected in Hungary in 1896, shortly afterwards in other countries—1900 in England, 1908 in Germany, 1909 in Newfoundland, 1912 in Sweden, etc. Almost everywhere the disease has retained its hold and spread, but not in Sweden, where since the thorough disinfection of the soil with 1 per cent. formalin in the years 1913 and 1914 (Eriksson, 1914; Hammarlund, 1915) it was not observed up to 1924, although present in Norway (1922), Denmark (1923), and Finland (1924). In 1928, however, the disease reappeared in Sweden in several places (Lindfors, 1928; Eriksson, 1928).

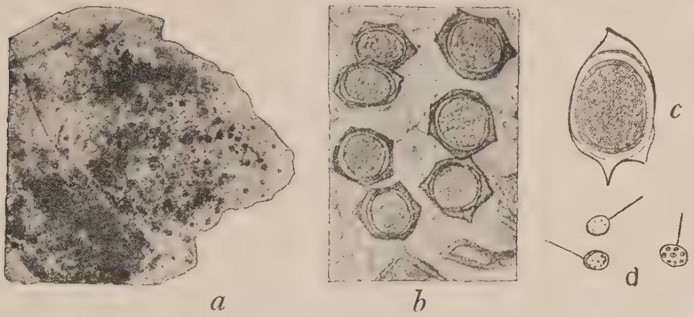

FIG. 42.—Fungus causing Wart Disease of Potato.

a, section of wart showing sporangia (slightly magnified); *b*, part of tissue with sporangia (highly magnified); *c*, mature sporangium; *d*, zoospores. (*a*, *c*, and *d*, after T. Johnson; *b*, after K. Schilbersky.)

Potatoes vary in their susceptibility to the disease; thus of some fifty varieties tested in England it was found that about one-third were immune. In Germany 30 per cent. of the varieties grown in Brandenburg were immune, 31 per cent. in Pomerania, 12 per cent. in Hanover and East Prussia (Spieckermann, 1923).

In North America, where the disease was observed not only in the east (Newfoundland, 1909; Pennsylvania, 1918) but also in some places in the central states, two methods of control have been devised: (1) The growing of immune varieties. After trials extending over a number of years some twenty-six varieties were regarded as immune and recommended for seed: in 1921 upwards of 10,000 bushels of Spaulding Rose were distributed to growers by the Pennsylvania Department of Agriculture. Other approved varieties are Green Mountain, Irish Cobbler, Ehnola, McCormack, etc. (2) The isolation of infected areas by a zone in which only immune varieties may be grown (Hartman, 1923).

In England, the Ministry of Agriculture and Fisheries has undertaken the testing of varieties, and has recommended as immune a certain number, such as Dargill Early, Snowdrop, King George, Great Scot, Tinwald Perfection, Kerr's Pink, Majestic, etc. A detailed list is published by the Ministry each year.

Preventive and Control Measures :

(1) All diseased potatoes, together with the haulms, must be destroyed at once, and the potatoes from an infected field must not be used for seed even though they appear to be healthy. (2) On fields where the disease has appeared, only immune varieties may be grown. (3) Great care must be taken that the soil from an infected field is not carried by men, animals, or implements to a clean field. (4) In Sweden, disinfection of the soil with 1 per cent. formalin solution is considered to have been successful. (5) When seed is bought it should be carefully examined for traces of disease. (6) Any potatoes under suspicion of being diseased should not be fed to stock unless boiled or steamed. Such potatoes must not be thrown on the manure heap.

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Blight of Flax

Olpidiaster radicis (de Wild.) Pasch.

This disease, known in Belgium, Holland, and France as "Brulure," appears in early summer in patches on flax plants in the field, and causes the stems of the plants to wilt and droop over. The outermost branches of the root become glassy in appearance and easily break off. In wet weather or on water-logged soil the disease spreads rapidly over the whole field.

The fungus causing the disease was originally named *Asterocystis radicis* de Wild., but as this name had already been used for a genus of Algæ the name *Olpidiaster radicis* (de Wild.) Pasch. was adopted.

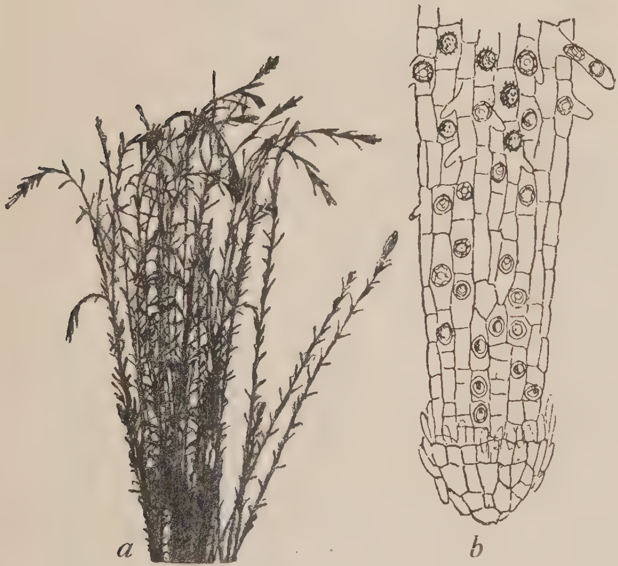

FIG. 43.—Blight of Flax.

a, infected plant in early stage of disease, tips drooping ; *b*, tip of a root branch with fungi in the cells. (*a*, after E. Marchal ; *b*, after V. Ducomet.)

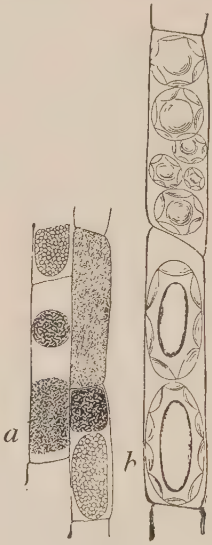

FIG. 44.—Blight of Flax.

a, cells from outer parenchyma of root with fungi inside the cells (early stages) ; *b*, cells containing resting spores. (After E. Marchal.)

The fungus lives in the surface cells of the small lateral roots, and finds entrance to the root just above the tip, usually when the plants are from 13–18 days old ; when a plant is 25 days old infection can no longer take place. The fungus spreads from cell to cell, and in the affected cells the plasmodium appears to contain protoplasm of the cell and of the fungus, and so may be regarded as "Mycoplasm" (see p. 74). This plasmodium is very dense in appearance and sometimes fills the whole cell; it is gradually changed into a mass—or several masses—of zoospores which escape through a hole in the cell wall. The fungus lives on from year to year by means of resting spores, one or more of which are present in each cell. The fungus also infects the roots of other plants, such as cabbage, turnips, radishes, spinach, peas, clover, and lucerne. The disease is likewise known in Germany and Ireland.

The only measure that can be taken against the disease is to abandon the growing of flax on an infected field for 7–10 years (Arnaud, 1914).

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The following fungi, belonging also to the Chytridiaceæ, produce diseases on agricultural plants: **Urophlyctis Alfalfæ** (Lagerh.) Magn. causes a disease known as "Crown Gall of Lucerne," which attacks the base of the root and gives rise to numerous coral-like growths about the size of a pea. The disease was first observed in South America; later in Switzerland, Italy, Germany, England, Holland, the United States, etc. In some places the disease has caused considerable damage to lucerne crops (Lagerheim, 1898; Magnus, 1901, 1902; Arnaud, 1916; Jones

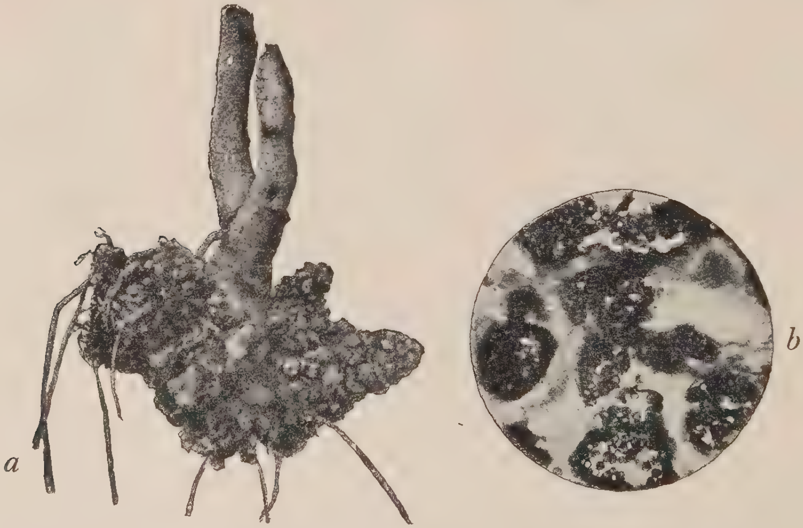

Fig. 45.—*Urophlyctis Alfalfæ.*
a, root of lucerne with galls; *b*, section of a gall, numerous cavities filled with resting spores. (After G. Korff.)

and Drechsler, 1920). **U. leproides** (Trab.) Magn. gives rise to large, short-stalked, irregularly furrowed outgrowths on the upper or middle portions of the root of the sugar beet. In section, the outgrowths show numerous dark dots. The disease has been observed in Algiers, France, and other countries (Vuillemin, 1906, 1917).

Olpidium Brassicæ Wor. attacks young cabbage plants in the spring, especially if they are crowded and have been kept very moist. The base of the root turns black, the stalk breaks off, and the plant dies. The disease is known as " Seedling Blight " (Woronin, 1878).

Olpidium Trifolii (Pass.) Schröt. and **Urophlyctis bohemica** Bub. give rise to blister-like pustules on the leaves and petioles of species of clover (Bubák, 1902).

Pyroctonum sphæricum Prun. has appeared with injurious effect on wheat in the south-west of France. The plants cease to grow, become yellow, and large, dead patches occur in the wheat-fields (Prunet, 1894).

Rheosporangium aphanidermatus Eds. attacks seedlings of sugar beet and radishes in the United Kingdom. The symptoms are similar to those caused by *Pythium de Baryanum* Hesse, *Aphanomyces lævis* de Bary, etc. (Edson, 1915).

CHAPTER IV

PERONOSPORACEÆ—DOWNY MILDEWS

THE fungi are almost exclusively parasitic. They have a well-developed and much-branched mycelium. The vegetative part of the mycelium, that is the part concerned in nourishment, ramifies between the cells of the host plant and penetrates into the cells themselves by short side branches (haustoria). The fructifying, *i.e.* the reproductive, cells (spores) are produced outside the host plant in the form of a grey covering or felt. Under the microscope this felt is seen to consist of numerous, erect aerial hyphæ (conidiophores) which are usually branched, and from the ends of which aerial spores, or conidia, are abstricted; it is by means of these spores that the disease is disseminated.

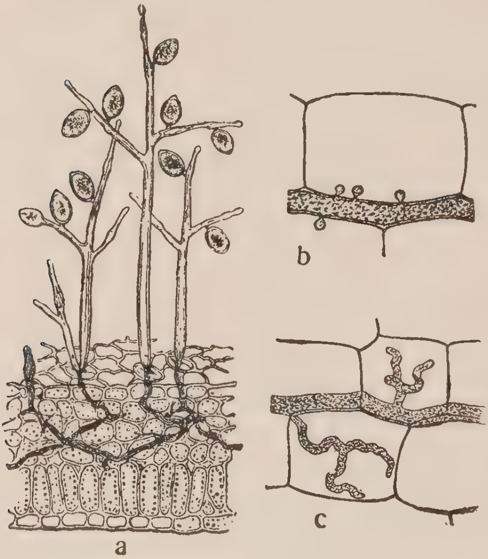

FIG. 46.—Mycelium of *Peronosporaceæ*. *a*, section of a diseased potato leaf, internally vegetative mycelium, externally condiophores with conidia; *b*, mycelium with short spherical haustoria (*Cystopus*); *c*, mycelium with long branched haustoria (*Peronospora*).

The germination of the conidia takes place in two ways: either the contents of the conidium divides up into a number of zoospores, each of which puts out a germ- or infection-tube, or else the conidium produces a single germ-tube. Sometimes, as in the species *Pythium*, there is a third form of reproduction in which the contents of the conidium is extruded into a thin-walled vesicle and afterwards divides into numerous zoospores. The conidia are either produced on much-branched conidiophores (as in the genera *Phytophthora* and *Peronospora*) or else in long bead-like chains (in the genus *Cystopus*).

The fungus over-winters by means of thick-walled resting spores (oospores) which are formed within the host tissues in the later part of the year when decay is about to begin; these oospores germinate in the following spring. In some cases, *e.g. Phytophthora infestans* and *Peronospora Spinaciæ*, it has been stated that the protoplasm of the fungus and that of the cell pass the winter in union, and that from this symbiosis the fungus may, in a certain stage of development of the host plant and under certain external conditions, free itself and produce a parasitic mycelium. (The "Mycoplasm" theory, see p. 74.)

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"Damping-off" Disease

Pythium de Baryanum Hesse.

This fungus lives in moist soil which is rich in humus, and it is found especially in seed beds from whence it passes to young seedlings belonging to various plant families. It not only attacks the stem below the cotyledons and the upper part of the root, but it can also attack and kill the tip of the main root, as well as the young lateral roots, during the whole growing period of the plant. The fungus may, in fact, enter the seed and destroy the young seedling before it has reached the surface of the

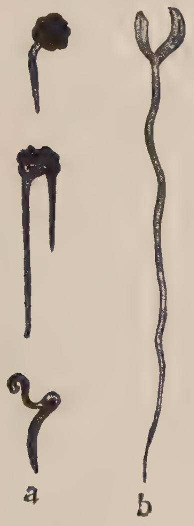

FIG. 47.—Mangel Seedlings attacked by *Pythium de Baryanum*.

a, diseased seedlings; *b*, healthy seedling of same age. (After W. Busse.)

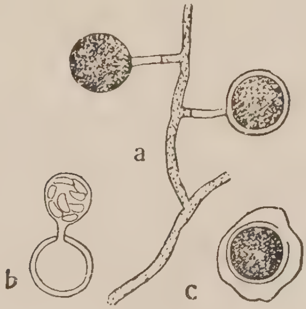

FIG. 48.—*Pythium de Baryanum*.

a, hyphæ with sporangia; *b*, sporangia about to produce zoospores; *c*, resting spore. (After E. Rostrup.)

soil. The infected tissues of the growing seedling take on a glassy, yellowish appearance, lose their turgidity, dry up, and become discoloured. The small seedling plants become flaccid, bend over and die. In a moist atmosphere, short hyphæ grow out from the surface of the diseased organs, and it is from the tips of these hyphæ that spherical outgrowths are produced. These sporangia may germinate at once, in which case the protoplasmic content is extruded and forms a vesicle, afterwards dividing up into numerous zoospores. There is an alternative method of germination in which, instead of sporangia with zoospores, conidia are formed, which germinate by a single tube. Towards the autumn, resting spores are produced as the result of a sexual process, and by this means the fungus is carried over the winter. These spores are formed in the decaying plant tissues and pass with them into the soil. Moist, warm conditions favour the spread of the disease, as does also the crowding of the seedlings in the seed bed.

The fungus attacks the seedlings of many kinds of plants, such as turnips, cabbage, lupine, asparagus, pea, barley, stock, etc. More rarely the older parts of plants, such as the tubers and stems of the potato, are attacked by the fungus.

A number of other species of fungi are able to produce diseases in seedlings similar to the one described above. The most important of these are : *Phoma Betæ* Frank and *Aphanomyces lævis* de By. (Peters, 1913), and then species of the genera *Arthrobotrys* (v. Tubeuf, 1902), *Gibberella* (Dickson, 1923), and *Fusarium* (Appel, 1924).

It may be mentioned that some investigators consider that the fungus is not the cause of the disease, but that the weakness or ill-health of the plant is responsible for the outbreak. The best means, therefore, of preventing the disease is not to proceed against the fungus, but to take steps to increase the vitality and power of resistance of the plant (Karlson, 1891).

Preventive and Control Measures :

(1) The upper layer of soil in the boxes or frames in which seedlings are raised should be mixed with fine sand, and the temperature and moisture conditions must be moderate during the early stages of growth. (2) If the disease has appeared in the seed bed in the previous year, new soil should be procured and the woodwork and glass should be carefully cleaned. (3) Very wet soil must be drained, and such operations as early ploughing after harvest, repeated loosening of the soil with the hoe, and adequate manuring with nitrogenous and phosphatic manures are desirable. (4) If there is a possibility that the disease may be carried in the seed, the latter should lie in a moist condition for two or three days, and then be soaked for two hours in a solution of copper sulphate (1–2 per cent.) before being sown.

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Late Blight of Potato

Phytophthora infestans (Mont.) de By.

This much-discussed disease of the potato plant appears in the field in late summer, in normal years about the middle of August, sometimes two or three weeks earlier or later. The disease first manifests itself by the appearance of large, dark spots on the deep green leaves. From day to day, these dark-coloured patches increase in size and number until finally, at the end of one to three weeks (according to the prevailing weather), the whole of the foliage is black and dead, and the stems stand bare. On approaching a field in which there is a diseased crop, a characteristic, unpleasant smell is noticed.

In addition to the late attack, as given above, an early form of outbreak was observed in Sweden in 1910, 1911 and 1918 on potato plants grown in hot beds under glass. When the seed potatoes were planted in hot beds in January or February and the soil kept very moist, the

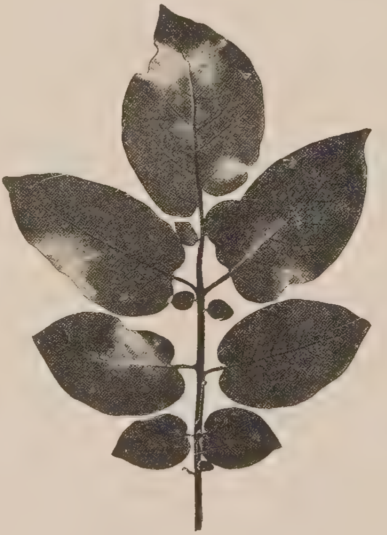

FIG. 49.—Primary Outbreak of Potato Blight in late Summer. (Orig.)

disease appeared suddenly about the middle of April, at the time when the fully-grown plants were beginning to develop new tubers. This early attack of blight did not appear on the leaves, which almost without exception remained healthy, but on the main stem and its branches in the form of long, dark, downy streaks, and also on the leaf-stalks, which as a result of the disease remained as thin as a needle.

In both cases—the late outbreak in the field and the early appearance in the hot bed—there is apparently the same phase of the life-history of the causal fungus, *Phytophthora infestans* (Mont.) de By. Although there are differences in the external characters of these two forms there is a noticeable biological similarity between them in that they both attack the potato plant when it has reached the same stage of development, *i.e.* when the parts above ground have attained their maximum growth

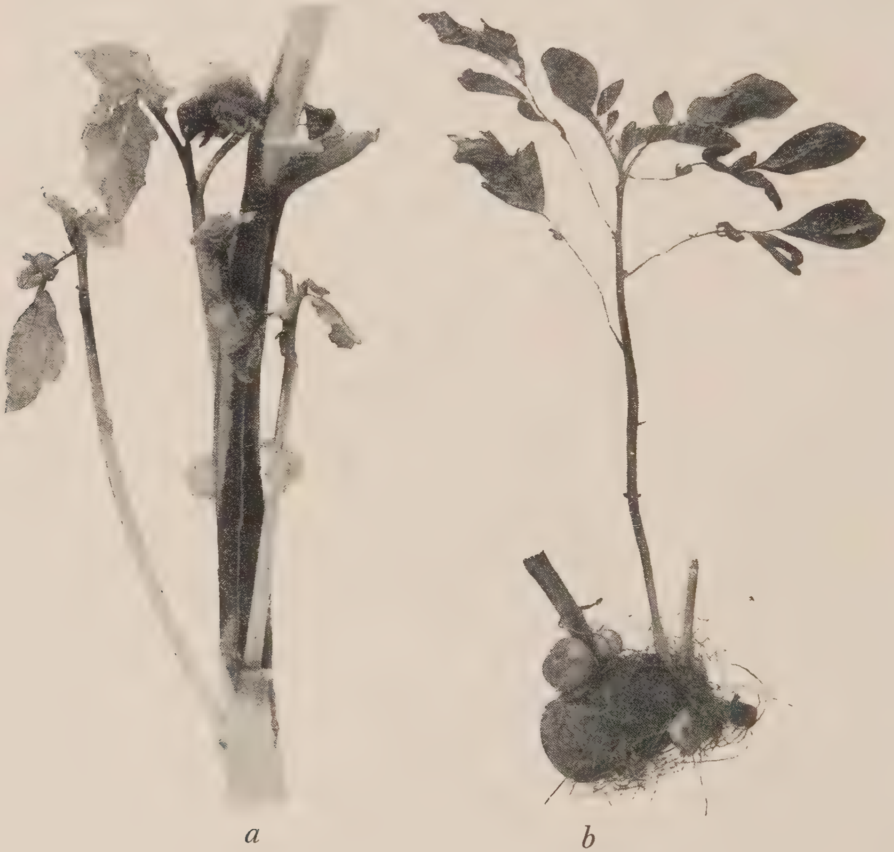

Fig. 50.—Early Primary Outbreak of Potato Blight in Hot-bed. *a*, long dark streaks on stem; *b*, thin, diseased, leaf-stalks. (Orig.)

and the plant is about to form tubers. This phenomenon points unquestionably to there being some infective substance within the plant, and makes it easier to understand the regularly late appearance of the disease in the potato fields.

It becomes, therefore, important to enquire : How this disease first reaches the foliage of the potato plant, and how the fungus over-winters.

Since the appearance of potato blight in Europe in 1845, the mode of hibernation of the fungus has been much discussed. About 1875 opinions on this point were widely divergent, some scientific workers claiming to have observed winter spores (oospores) in certain parts of the plant (W. G. Smith, Berkeley, Vize, Plowright). Other workers, however, maintained that the oospores belonged to a closely-related fungus of the genus *Pythium*, and were accidentally present in the plant (de Bary, Brefeld, Magnus). To decide this question, de Bary, at the invitation of the Royal Agricultural Society of England, undertook an investigation and published his conclusions in 1876. He upheld his previous opinion, and as this was generally accepted, the question of the over-wintering of the fungus remained for a long time unsolved.

In spite, however, of de Bary's views, differences of opinion continued to exist and further investigations were undertaken in the following decade. In addition to the older views that the disease exists (*a*) in the tuber or in the shoot growing from it (de Bary), (*b*) in a perennial mycelium in the soil (Kühn), (*c*) in the oospores found in the decayed portions of the plant (W. G. Smith), a number of fresh suggestions were made. One of these was that a spore-forming mycelium is produced on the surface of the tuber and passes to the foliage (Hecke, 1898); another that the latent fungus exists throughout the tissues of the potato plant in an unrecognisable granular or mucilaginous state (W. G. Smith, 1884 ; Wilson, 1891 ; Massee, 1906).

Finally, at the beginning of the present century, a new period of activity began in the investigation of the disease, and comprehensive researches were undertaken in the United States and in England (Clinton, 1904–10 ; Jones, Lutman and Giddings, 1904–12 ; Pethybridge and Murphy, 1911–13 ; Melhus, 1913–15). The chief method used in these investigations was the cultivation of the fungus on artificial media, and by this means it was found possible to produce rudimentary oospores artificially. In spite, however, of all efforts the question of the over-wintering of the fungus remained unsolved.

In order to investigate the hibernation of the fungus and the reappearance of the disease in a succeeding year, experiments were commenced in Sweden in 1915. These experiments were on different lines from those followed in the English and American researches. As a starting-point, the primary large spots which first appear in late summer on the leaves were chosen, and a very careful microscopical examination was made of the changes which take place in the formation of these spots, the object being to ascertain the origin of the mycelium.

On the under side of the leaf-spot it is possible, with the aid of a lens, to distinguish several zones which merge into one another. Thus (*a*) a middle zone which is brown or black, almost dead ; (*b*) a grey felted zone ; (*c*) a pale green zone ; (*d*) a bright green zone. The detailed structure of the individual zones was examined in a large number of microtome preparations which were stained according to modern cytological methods.

The first important result was the discovery of oospores in the disorganised spongy tissues of zones (*a*) and (*b*). The spores, which were either separate or in groups of 2–3, were spherical with a smooth thick wall ; the contents granular with a number of nuclear bodies. In the same zone in which the spores were found it was possible to follow their development. Germination took place at once, so that the spores were true summer spores and not resting spores. They germinated either as conidia with germ-tubes and entered a neighbouring stoma, or as sporangia, the contents of which divided up into spores each of which put out a germ-tube and could penetrate a stoma. From the hyphæ, an aerial mycelium, characteristic of this fungus, developed, and from its branches spores were formed which developed further as zoosporangia.

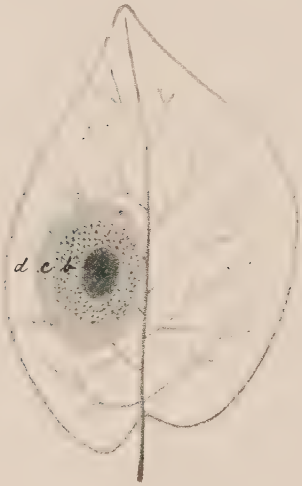

Fig. 51.—Primary Outbreak on Potato Leaf, showing the Various Zones in the Spot. (Diagrammatic.)

The stages of development prior to the formation of the oospores can be followed in the peripheral portions of the leaf-spots. In certain cells of the zones (*c*) and (*d*) were noticed numerous small, punctated, granular bodies which gave a peculiar, reticulate structure, different from that of normal protoplasm, to the cell contents. These peculiar granular bodies are the fungus-element in the symbiosis between the protoplasm of the cell and that of the fungus, the so-called "Mycoplasm," and the condition just described can be regarded as the resting stage of the mycoplasm.

The succeeding stages in the life of the mycoplasm can also be observed in preparations made from the respective zones : the dissolution of the chlorophyll granules ; the genesis of a number (3–6) of nucleoli in the protoplasm and the aggregation of certain portions of turbid protoplasm—the ripening stage of the mycoplasm—until finally the turbid aggregates of the mycoplasm pass through the surrounding cell wall and give rise, in the intercellular spaces, to a plasmodial strand which develops into a true mycelial hypha. Thus there is an intercellular mycelium which is ready to form male and female branches (antheridia and oogonia) ; by the copulation of these organs the oospores are formed.

In what way the fungus penetrates in the plasmal form into the potato plant, and particularly into the newly-formed stolons and young tubers, has not yet been discovered.

The disease spreads very rapidly, both in the hot bed in spring and in the field in autumn, by means of the conidia which are produced, in the manner described, at the time of the primary outbreak. These primary spores (conidia) develop as sporangia. The turbid contents of the sporangium divides into a number of zoospores which pass out through a small hole in the sporangium; the zoospores are biciliate and swim actively. After half an hour or so, movement ceases, the cilia are lost, the zoospores germinate and quickly produce secondary patches on any leaves that they infect. From these secondary patches a generation of secondary conidia arises.

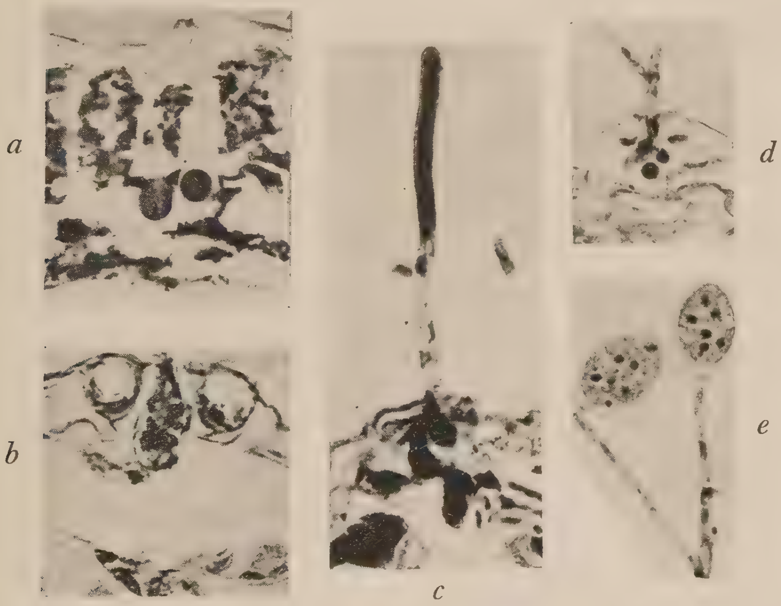

FIG. 52.—Development of the Fungus in the Primary Outbreak.

a, disorganized leaf tissue with two oospores; *b*, germinating oospore, closely applied to cells of stoma; *c*, simple germ-tube with rudimentary branch; *d*, germ-tube with side branch: two ungerminated spheres, from the oospore, in respiratory cavity; *e*, two conidia which develop as zoosporangia (primary conidia). (Orig.)

As to the way in which these secondary spores germinate nothing certain is known. Since the middle of the last century it has been known that these spores—which are generally known as conidia—can germinate in two ways, either as sporangia with zoospores or as conidia with a single germ-tube. The reason for this difference in the method of germination has not yet been explained, although much has been written about it. It is possible that the conidial germination is a property of the secondary spore generation; if so it might be expected that there would be differences in the life-history and function of the two kinds of spores.

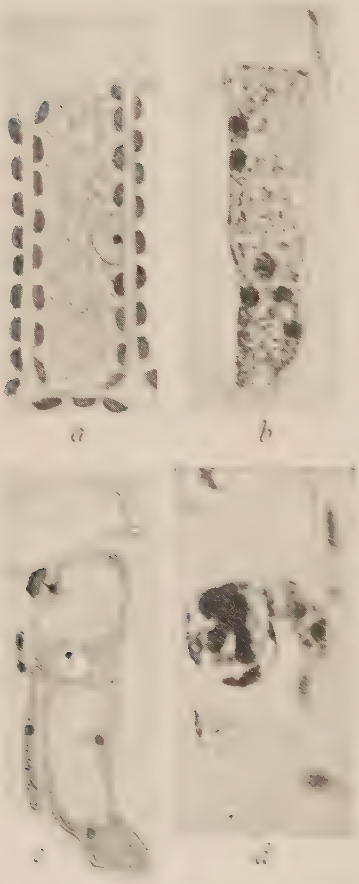

Fig. 53.—Previous Life of the Fungus within the Leaf.

a, cell from green portion of leaf (zone d, Fig. 51) with mycoplasm at rest; b, cell from light green portion (zone c) with mycoplasm in ripe nucleolar stage; c, cell with two extrusions of plasm into the intercellular spaces; d, copulation between an antheridium (right) and an oogonium (left). (Orig.)

The spread of potato blight depends upon the weather at the time of the first outbreak of disease or shortly thereafter. In moist, or more particularly misty, weather the disease spreads very quickly. Within a few days the characteristic spots are to be found on every plant, and after one or two weeks the whole field is black. If, on the other hand, the weather is very dry at the time mentioned above, the disease spreads but slowly and may even cease to develop.

When the potato haulms are withered in the manner already described it is practically certain that the young tubers which are developing will become infected. The disease manifests itself in the tubers by the dark, generally brown, patches which sink more or less deeply into the flesh of the potato. The view generally taken is that the disease reaches the tubers from the soil, the fungus being washed down by rain. Experiments made in England and Denmark in the eighties of the last century support this view, because it was found that the spores could be held back in a layer of sand of sufficient thickness. The benefit of "earthing up" potatoes as a means of checking the disease is well known (J. L. Jensen, 1884; Plowright, 1884).

The above explanation as to the manner in which the disease is transmitted to the tubers was not supported by trials made in Germany, as in them it was not found possible to infect healthy potatoes from diseased haulms (Wehmer, 1898). In almost all of these trials the results were negative, and even when the potatoes became diseased it was shown that they had not been attacked by the "blight" fungus, but by a bacterial rot. It would appear, therefore, that the disease reaches the tubers in a manner similar to that described above in the case of leaves; that is to say, after undergoing certain changes in the tuber, probably in the peripheral tissues, the fungal plasm gives rise to an intercellular mycelium which produces the disease. Certain early observations, which have now been forgotten but not refuted, are in favour of this view (W. G. Smith, 1876).

The appearance of potato blight in Europe is generally stated to have been in 1845 when the disease was described and named. In the same year it was found to be spread over large areas in many countries. The rapid spread of the disease was attributed to the weather conditions in the summer and autumn of that year. A satisfactory explanation for the appearance of the disease has not been furnished, and equally unexplained is its sudden and general outbreak at the same time in North America; it was first found in Australia in 1904 (McAlpine).

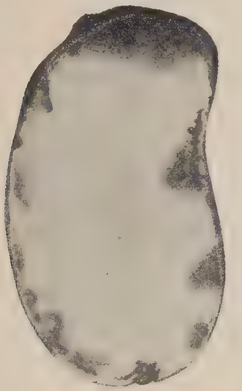

FIG. 54.—Young Potato Tuber attacked by *Phytophthora infestans*. (After O. Appel.)

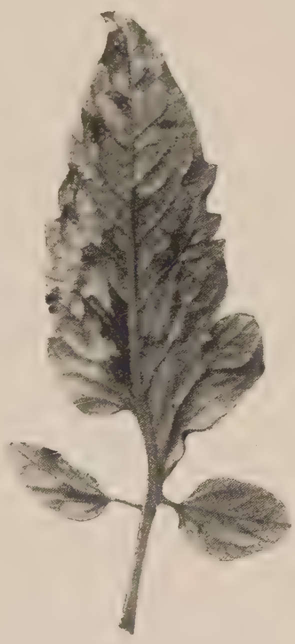

FIG. 55.—Tomato Leaf attacked by *Phytophthora infestans*. (Orig.)

The fungus has also been found on plants more or less closely related to the potato; for example, on *Solanum tuberosum*, *S. caripense*, *S. stoloniferum*, *S. utile*, *S. maglia*, *S. verrucosum*, *S. Dulcamara*, *S. laciniatum*, *S. marginatum*, *S. muricatum*, *S. tuberosum* × *utile*, *S. Lycopersicum*, *Anthocercis viscosa*, *Petunia hybrida*, *Schizanthus Grahami*, and *Datura Metel*.

Experience has shown that some varieties of potatoes are not readily attacked by this downy mildew, and use has been made of the property in controlling the disease. As every grower of potatoes knows, the resistance of a variety may alter under different conditions and may not be constant. Further, resistance is not the only factor to be considered; properties such as time of ripening, cropping power, taste, starch-content, etc., have all to be taken into consideration.

It was assumed for a long time that the fungus described above was responsible for "Wet Rot" and "Dry Rot" of potatoes. It has been already mentioned, however, that these diseases are caused by bacteria.

Preventive and Control Measures:

(1) Only healthy tubers must be used for seed. Potatoes from infected fields should be discarded, even though they appear to be healthy, as there is every reason to think that the disease is mainly kept alive from year to year in slightly diseased tubers. (2) The ground should be suitable; a well-drained sandy soil or a loam containing a large proportion of sand is best. (3) Disease-resistant varieties should be grown, and local experience may be used as a guide in selecting the best varieties. (4) Potatoes should not be grown year after year on the same field. (5) Dung should not be applied directly to the crop, certainly not fresh dung. (6) Potatoes should be grown on high ridges and the soil "earthed up" round the plants so that the tubers are protected by a thick layer of soil, some 3½ inches in the case of a sandy soil and 4–5 inches in a loam. This high "earthing up" requires the plants to be placed a wider distance apart; the rows, in the case of a sandy soil, should be about 2½ feet from one another, and on heavier soil about 3 feet. (7) The plants should be sprayed as soon as the spots are noticed on the leaves, or, better still, before they appear, if the time at which the outbreak usually occurs can be learnt from local experience. Bordeaux mixture (1 per cent. strength) should be used and the spraying should be repeated after 3–4 weeks. The time of spraying will vary according to the locality and the variety of the potato. Spraying with

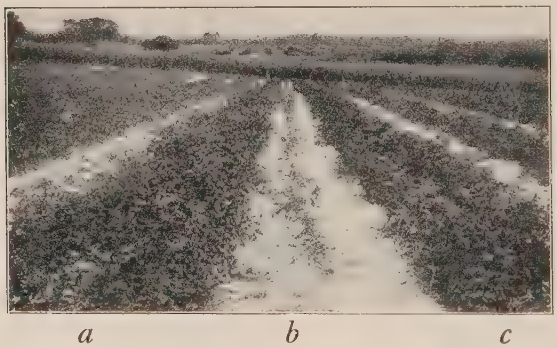

FIG 56.—Potato Crop sprayed with Bordeaux Mixture. *a*, sprayed every second week; *b*, unsprayed; *c*, sprayed three times. (After B. M. Duggar.)

Bordeaux mixture causes the foliage to remain green for a longer period (1–5 weeks), and it increases the number of tubers that can be stored as well as the total crop. (8) Potatoes should not be harvested too early, as the air at that time is filled with infective material from partially withered haulms. (9) The potatoes must be stored in a dry condition, and this checks the subsequent development of disease in the stored tubers.

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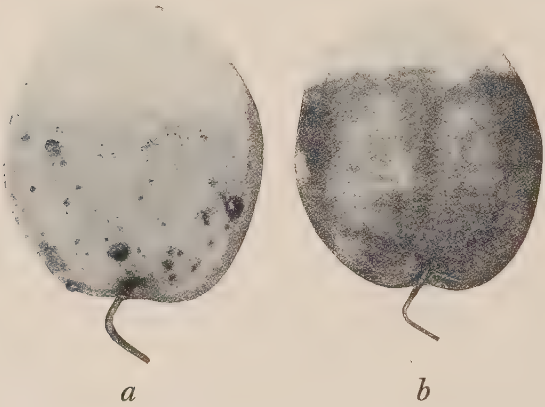
Pink Rot of Potato

Phytophthora erythroseptica Peth.

This disease differs in several important characteristics from "Blight." In the case of early potatoes, the disease may appear on the haulms about the end of July. The leaves become yellowish-green, then yellow with incurved margins, and finally dry and crinkled. A section of the diseased stem shows the vascular bundles to be discoloured. When the infection is very severe the haulms collapse. The chief seat of the disease is in the tuber, the attack beginning in the basal part or "heel" end and spreads from there to the "rose" end. In the early stages of the disease a potato when cut lengthwise shows no sharp division between the diseased and healthy tissues. After a few minutes' exposure to the air, the cut surface at the " heel " end turns pink, and after some hours the colour changes to brown and then to nearly black. The pink colour is also seen when potatoes suffering from the disease are injured during lifting, this symptom characterizes the disease.

An infected tuber is of a spongy, somewhat elastic, consistency, and if squeezed exudes a watery, acid juice. Such a tuber decays completely (" wet rot ") in a few days if kept at room temperature under a bell jar, producing a faint, acid odour similar to that of formalin solution. If the diseased tuber be left uncovered it becomes dry and withered, and so passes into the " dry rot " state.



[Fig. 57.—*Phytophthora erythroseptica*, diseased tubers. *a*, external appearance ; *b*, internal appearance ; red colour develops on diseased portion after a few hours' exposure to air. (After G. H. Pethybridge.)

In the soil, the decayed tuber produces a larger or smaller number of oospores below the skin, and these finally pass into the soil ; the germination of the oospores has been observed.

This disease was first noticed in 1909 in the west of Ireland ; two years later it appeared in the north of Ireland, and in 1915 in Holland. On fields where potatoes are grown year after year the disease becomes most serious, as much as 33 per cent. of the crop being infected. The spread of the disease by the tubers has not been observed. In Holland the fungus has been found parasitic on *Atropa Belladonna* (Westerdyk and van Luijk, 1920).

As a preventive measure it is recommended to dig up all diseased parts of the plant, to destroy them, and as far as possible avoid growing potatoes on the same soil year after year.

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Damping-off, or Foot Rot of Tomato

Phytophthora cryptogea Peth. & Laff.

This disease shows itself on tomato seedlings when they have reached the height of 4–6 inches; the outer tissues of the stem, immediately above the soil, become brown to black in colour, then shrivel and decay. For a time, nothing unusual is noticed on the other parts of the plant above ground, and they remain green and turgid; but gradually the leaves begin to curl, then become yellow, and finally die. If a diseased plant be drawn from the soil it is found that the rot in the peripheral tissues extends to the underground parts of the stem and to the root. Generally the primary root decays completely and secondary roots often arise from the healthy stem just above the diseased parts, enabling the plant to continue to live.

When an infected plant with root and lower part of stem is placed in water a mycelium with fructifications arises on the diseased portions. These fructifications are ovoid and behave either like sporangia and develop numerous zoospores, or they germinate as conidia by a single germ-tube. Oospores have not been found in the dead tissues, but oospore-like bodies have been produced in artificial cultures.

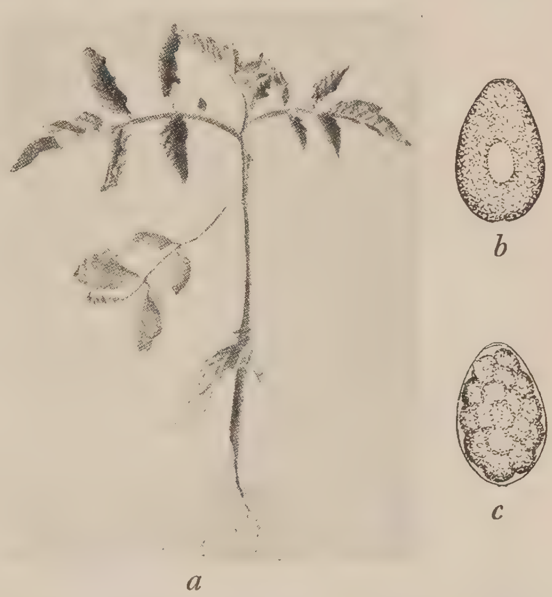

Fig. 58. --Damping off, or Foot Rot of Tomato Seedlings. *a*, diseased seedling; *b*, sporangia. (After G. H. Pethybridge and H. A. Lafferty.)

This disease was first observed in a market garden near Dublin (Ireland). In the first year only a few plants were attacked, in the second year about half of the crop, and in the third year almost all the plants died; at the same time the fungus appeared also in neighbouring gardens.

In the above-mentioned places the same fungus was observed on species of the genera *Cheiranthus*, *Aster*, and *Petunia*, and by artificial inoculation it was transmitted to *Solanum tuberosum*, *Gilia tricolor*, and *Fagus silvatica* as well as to green and ripe tomatoes. The fungus appears to live in the soil from one year to the next.

Preventive Measures :

(1) Tomato seedlings should be grown in soil that has been sterilized by heat. (2) Soil in which infected plants have grown should be removed. (3) Pots, boxes, and tools should be disinfected with 2 per cent. formalin solution. (4) If only a few plants are diseased, the infected parts may be removed and the remaining healthy portions be treated as cuttings. (5) All diseased plants that are not treated in the manner just described must be removed and burnt.

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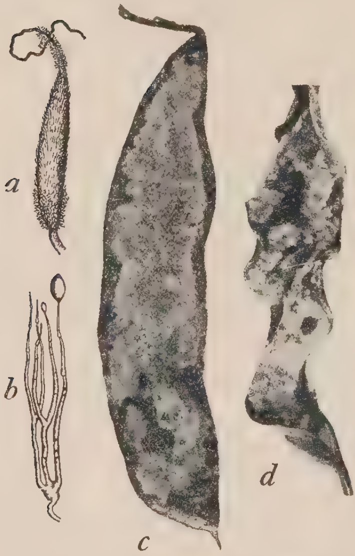

Fig. 59.—*Phytophthora Phaseoli.*

a, ovary with fungus at base and tip ; *b*, branched mycelium with sporangium ; *c*, fully-grown pod covered with felt of fungus ; *d*, shrivelled and distorted pod. (*a*, after W. C. Sturgis ; *b*, after K. Thaxter ; *c*, after B. D. Halsted ; *d*, after C. P. Clinton.)

Phytophthora Phaseoli Thaxt. This fungus attacks the Lima, or moon, bean (*Phaseolus lunatus*), producing Downy Mildew or Rot. The first appearance of the disease (from the middle of July to the middle of August) is a delicate, white felt of hyphæ on the young pods, at first mainly at the base and tip of the pod. The hyphæ are very long and only branched at the base ; each hypha bears several nodular swellings. From the tips of the hyphæ oval reproductive cells are formed, which either germinate directly as conidia or, as is generally the case, produce numerous (fifteen) zoospores which develop germ-tubes. On the mature pod the fungus produces large, white, irregularly-shaped patches which frequently almost cover the whole pod. In severe cases the pod shrivels up and the beans become useless. The young leaves, parts of the stem and flowers are also attacked by the fungus, and become misshapen and twisted. Wet weather in July and August increases the intensity of the attack very considerably, and in very wet years 50–90 per cent. of the crop has been lost in 3–4 weeks ; in dry seasons the losses are unimportant. The way in which the disease is carried on from year to year is not clear ; for a number of years attention was paid to the oospore stage, but it was only on one occasion (September, 1905), after a very wet summer, that oospores were isolated from the diseased seed and germinated in the following year (Clinton, 1906). The finding in one year only of the oospores does not satisfactorily solve the question of the over-wintering of the fungus and the regular appearance annually of the disease. Nor does the germination of the spores in the following April agree with the usual primary outbreak in the field some three or four months after that date—generally in August.

The disease was first observed and described in 1889 in the neighbourhood of New Haven (Connecticut). In 1897 it was found in New Jersey and other parts of the United States, also in Tiflis; in 1904 in Delaware (Long Island), etc. (Thaxter, 1889, 1890, 1893; Speschnew, 1897; Halsted, 1898, 1901; Sturgis, 1898, 1899; Clinton, 1906, 1909; Adams, 1924; Cook, 1924).

Preventive Measures :

(1) Seed from diseased crops not to be used. (2) Grow the beans on well-drained soil and not too closely together. (3) Beans not to be grown on infected soil for 2–3 years. (4) Spray with Bordeaux mixture 2–3 times from the middle of July onwards.

Beech Seedling Rot

Phytophthora Fagi R. Hart.

This disease attacks young beech seedlings up to the time the first foliage leaves are fully formed. Either the young seedling is attacked immediately after putting out its rootlet, in which case the plant never grows above the soil but rots, or the development is normal and apparently healthy cotyledons are formed. Within a short time, however, dark green spots appear at the base of the cotyledons, on adjacent parts of the stem, and on the foliage leaves. In continued damp weather, and especially in shady places, the plant rots, whereas in dry weather the diseased parts dry up and become of a reddish-brown colour, the plant continuing to grow. The disease spreads centrifugally from a centre of infection, and the fact that plants near to footpaths suffer most, points to the transfer of infective material on the shoes of pedestrians, hoofs of animals, wheels of vehicles, etc. The

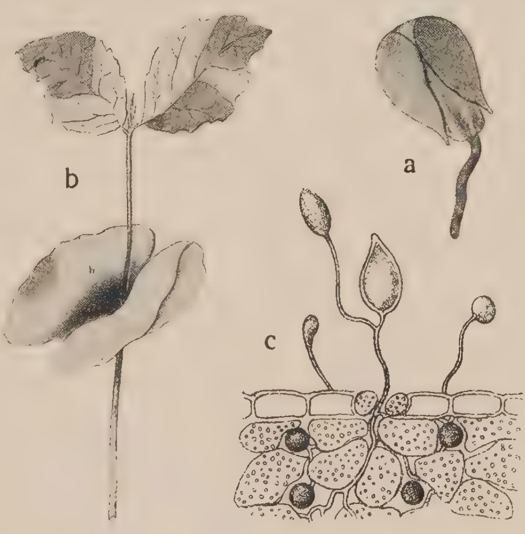

Fig. 60.—Beech Seedling Rot.

a, germinating seed; *b*, young seedling; *c*, epidermis of a diseased cotyledon. (After R. Hartig.)

epidemic lasts about four weeks, and the disease reappears in the same soil in the following year if beech seedlings are grown there.

On the margin of the diseased leaf, a greyish-white felt composed of hyphæ grows out from the tissue. Each hypha produces a reproductive cell at the tip which is pushed to one side as the hypha develops and another cell is formed. These cells are lemon-shaped and behave generally as sporangia, each cell developing ten zoospores as a rule, and from the zoospore one or more germ-tubes are put out. The reproductive cells can also function as conidia.

Oospores are formed within the infected cotyledons, leaves, and stems in very large numbers—it is calculated more than a million in a single plant. The oospores are smooth and colourless but later become yellowish-brown. Wet weather favours the formation of the oospores, and when the diseased parts of the plant decay the oospores pass into the soil and, it is assumed, lie dormant until, in the following year, they infect the young seedlings; the germination of the oospore has not, however, been observed. Water containing mycelium and oospores when applied to a seed bed caused 30 plants out of 8000 to be visibly attacked within a month, whilst ten days later all the plants were dead. The oospores remain viable in the soil and capable of causing infection for at least four years, and a handful of soil is sufficient to infect a large seed bed.

This disease was known in Germany as early as 1783, and since 1872 it has been reported from several districts in that country; it was first observed in Denmark in 1878.

Preventive Measures :

(1) Beech seeds should not be planted in old seed beds which have been used for the same crop for a number of years. (2) When single diseased plants appear among the healthy ones, the seed bed should be examined each day and all infected plants collected and buried in some remote place. (3) If the number of diseased plants is large the seed bed should be dug up. (4) The seed bed should not be in a shady place. (5) Workmen should keep off the seed bed as much as possible and avoid transferring infected soil, etc., on their shoes and clothes. (6) If mice are noticed they should be trapped or poisoned.

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A disease similar to the above has been observed—particularly in Denmark—on seedlings of species of *Acer*, *Fraxinus*, and *Pinus*.

Downy Mildew of Lilac

Phytophthora Syringæ Kleb.

This disease is seen in certain years on the shoots of forced lilac, and makes its appearance from the beginning of January onward. Some of the buds open abnormally or else shrivel and die, as do also the flower panicles which have already opened. Browning and shrivelling of the diseased parts of the plant are characteristic. In the case of small pot plants, the tip of the shoot dies back, whilst the buds lower down the branch produce a lot of leaf. When the disease attacks larger shrubs, growth ceases, sooner or later, in some of the twigs. Young flower panicles wither and fall. The centre of infection in such cases is in the lower part of the stem, generally just above the ground level, and the bark at this point is brown and shrivelled.

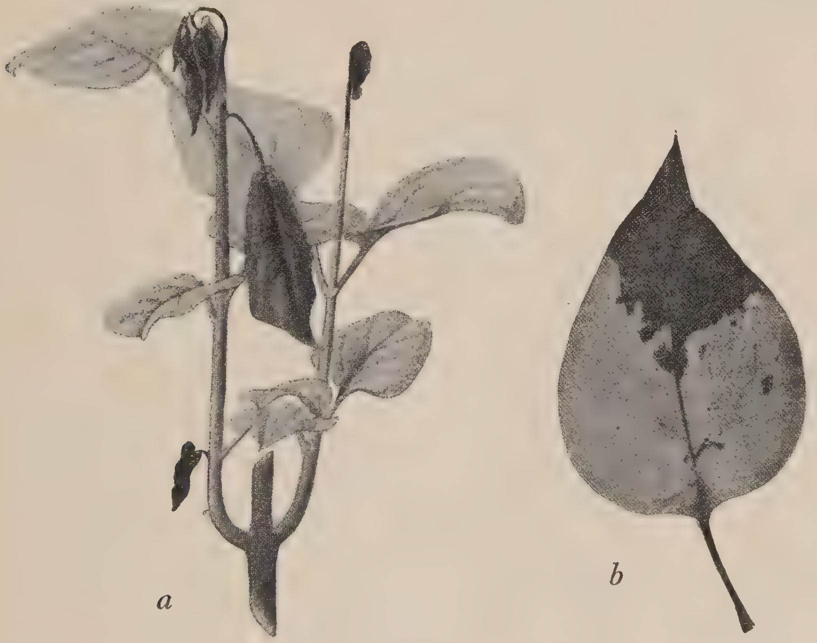

FIG. 61.—Downy Mildew of Lilac.
a, diseased twig ; *b*, diseased leaf. (*a*, after G. Arnaud ; *b*. after de Bruyn.)

Thick-walled oospores are found in large numbers in the brown portions of the tissues, especially in the leaves and flowers of diseased buds. The origin of the oospores and the part they play in the life of the fungus has not yet been adequately investigated. It may be surmised that the hypha-like mycelium which pushes out through the stomata arises directly from the oospores, and that from these hyphæ the aerial mycelium (which has been observed to be formed on germination in water) arises and produces sporangia by abstriction.

At different periods in the growth of the plant, after continued wet weather, brownish-black spots have been observed on the leaves. These spots have an irregular shape and are not limited by the leaf veins. On leaves bearing these spots, the fungal hyphæ push out through the stomata or else form oospores within the tissues. Infected leaves soon die and fall prematurely.

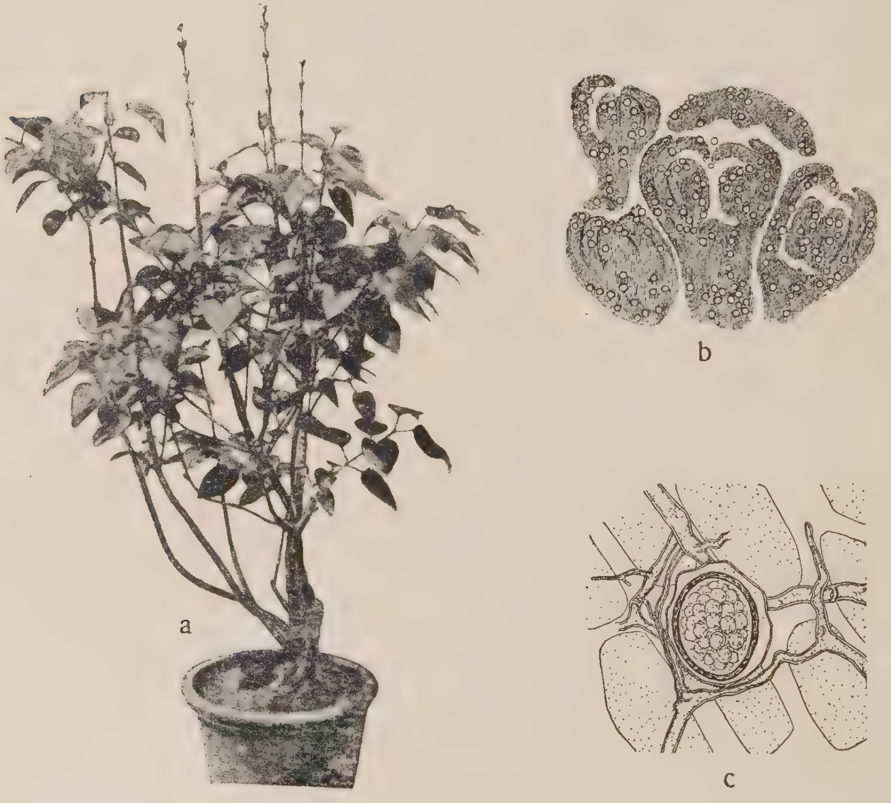

Fig. 62.—Downy Mildew of Lilac.

a, pot plant, tips of some twigs without leaves ; *b*, section of bud showing oospores ; *c*, mycelium with an oospore. (After H. Klebahn.)

It is assumed that this fungus lives in the soil as a saprophyte for the whole year, or for several years, and that unusually hot weather in August and September stimulates it to a parasitic mode of life. The first attacks of the parasite are on the lilac stem at ground level, entrance being through a wound, or on the tender leaf and flower structures. From these points the mycelium spreads through the cambium tissue and the vascular bundles. Artificial inoculations with pure cultures of the fungus have given positive results in the winter months, October to January (the development of the fungus reaching its maximum in January and February), but as soon as growth commences in the organs of the host the mycelium appears unable to develop further and dies down. It is true that from May to July oospores are met with, but they remain dormant until the winter.

The peculiar and characteristic mode of life of this fungus is possibly connected with its adaptation to the conditions which are connected with the forcing of the plants in winter.

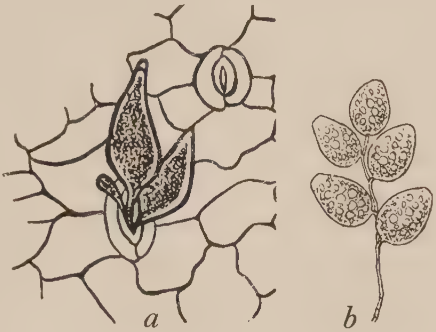

Fig. 63.—Downy Mildew of Lilac.

a, rudimentary sporangium pushing out of stoma; *b*, group of sporangia. (*a*, after de Bruyn; *b*, after H. Klebahn.)

The disease was mentioned as early as 1881 in England under the name of *Ovularia Syringæ* Berk. (Berkeley, 1881; W. G. Smith, 1883; Wilson, 1886); in 1905 it was examined in Germany and referred to its proper systematic place as *Phytophthora Syringæ* Kleb. There was an epidemic outbreak of the disease in 1913 in Holland (Aalsmeer) and again in 1919, in both cases after very wet autumn months. Probably in most countries in Northern and Mid-Europe, where forced lilac is grown, the disease is known.

By artificial inoculation the disease has been transmitted to *Ligustrum vulgare*, *Jasminum nudiflorum*, *Forsythia viridissima*, and *Cratægus Oxyacantha* with the formation of oospores, whilst shoots of *Pyrus communis* and *Prunus Cerasus* inoculated in a similar manner died without forming oospores. Attempts to infect species of the genera *Azalea*, *Betula*, *Carpinus*, *Fagus*, *Fraxinus*, *Juglans*, *Philadelphus*, *Platanus*, *Salix*, *Sorbus*, *Erica*, and *Calluna* were successful. Later investigations point to the lilac fungus being an independent species in spite of it being transmissible to other plants.

Quite recently a disease of *Pyrus Malus* due to this fungus has been observed in Ireland (Lafferty and Pethybridge, 1922).

Preventive and Control Measures:

(1) Destroy all diseased parts of plants by burning. (2) During the winter, the plants should be placed so that the buds are not too near the soil and are kept as dry as possible. (3) When handling the plants avoid causing any injury to the bark.

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Phytophthora Cactorum (Leb. & Cohn) Schröt. attacks *Cereus giganteus*, *C. speciosissimus*, *C. peruvianus*, *C. tephracanthus*, *C. martianus*, *Melocactus nigromentosus*, and *Echinops Euriesii*. An aerial mycelium which abstricts conidia is formed on the surface of the cactus stem, otherwise the epidermis is little affected. Within the stem, however, there are decayed portions containing oospores, the germination of the latter has not yet been observed. In 1868 the disease appeared in a glasshouse in Breslau ; recently it has been found in several glasshouses at the Berlin Botanical Garden (Lebert and Cohn, 1875 ; Lindau, 1908 ; Himmelbauer, 1911).

Certain diseases on other plants are ascribed to this fungus, *e.g.* on ginseng (*Panax quinquefolia*) in Japan and North America (Hori, 1907 ; Whetzel, 1912) ; on pears and apples in England (Cotton, 1921), in Holland (van Poeteren, 1922), and in North America (Rose and Lindegren, 1923) ; on tulips in North America (Stevens and Plunkett, 1925) ; and on rhubarb in North America (Beach, 1922).

Phytophthora omnivora de By. occurs on *Cleome violacea*, *Alonsoa caulialata*, *Schizanthus pinnatus*, *Gilia capitata*, *Fagopyrum marginatum*, and *Clarkia elegans*. At the beginning of the flowering period, the plants become brown and wilted from the soil level upwards and either decay or dry up. From the mycelium in the parenchyma of the cortex and leaves, branches pass through the stomata, and at the tips of the hyphæ sporangia are formed. Only in *Clarkia* and *Gilia* were oospores found which germinated in the cells of the parenchyma and epidermis. From oospores which ripened in August, germination occurred in November when the spores were kept in a warm room under water. When the spores were kept cool, even to freezing temperature in December, they germinated in December and January, some fresh germination being observed as late as April. By artificial inoculation the fungus was transmitted to *Œnothera biennis* and *Epilobium roseum*, where it formed sporangia as well as oospores. In the same way, infection was carried to *Lepidium sativum*, where only sporangia were developed and the plant was quickly destroyed; to *Salpiglossus sinnata*, where only sporangia were developed; and to the rosette of leaves on species of *Sempervivum*. The disease appeared destructively at Strasburg (de Bary, 1881) in 1878–79. Latterly the fungus has been found to produce rots on calceolarias, asters, and gooseberries (Osterwalder, 1905–10; Müller-Thurgau, Osterwalder and Jegen, 1922). A similar rot on strawberries has been observed in Switzerland. Here the disease first appears near, or above, the calyx and spreads to the fruit, destroying the colour and turning the berry brown. The flesh of the fruit undergoes a change, becoming rubber-like and firm. Oospores are formed within the fruit and in wet weather on its surface; sometimes sporangia are also found inside the berry. It is probable that in the soil of an infected strawberry plantation a large number of oospores are present, and they give rise to the disease in the following summer (Osterwalder, 1912).

Another Phytophthora rot on pears and apples was reported from Switzerland in 1906, Belgium 1908, and Bohemia 1910. The symptoms of the disease are a browning of the skin of the fruit, either in patches or over the whole surface, at the time of ripening or even earlier. The flesh below the brown skin is also discoloured to the depth of 1 cm. In moist surroundings, a white aerial mycelium arises on the diseased surface and produces unbranched conidiophores which abstrict ovoid or spherical conidia that germinate by a germ-tube. In the diseased tissue, globose oospores are produced. The flesh of the fruit remains hard and does not mellow (Osterwalder, 1906; Marchal, 1908; Bubák, 1910).

Finally, a disease known in Italy as "Cancrena pedale," which occurs on *Capsicum annuum* and *Solanum Melongena*, comes in this group (Trotter, 1924).

Phytophthora Sempervivi Schrenk. on *Sempervivum albidum*, *S. tectorum*, *S. glaucum*, and *S. stenopetalum*. The disease begins in the young inflorescences and spreads to the stems and leaves, producing a "rot." Sporangia arise on the surface mycelium and oospores are met with in the tissues of the cortex and hairs. The disease was observed in 1875 in the Leipzic Botanical Garden (Schrenk, 1875).

It has been suggested that the various forms of disease described under *Phytophthora omnivora* and *Ph. Sempervivi* should be placed under *Ph. Cactorum* (Himmelbauer, 1911).

Phytophthora parasitica on *Ricinus communis*. Circular, brown spots appear on the cotyledons of young seedlings, one spot on each leaf, and soon afterwards elongated patches are found on the leaf-stalk and stem. In consequence of the attack of the fungus, the leaf falls or the seedling dies—often 30–40 per cent. of the plants perish. On the fully developed leaves of older plants, large circular, yellowish-brown spots arise, and these, through the peripheral growth of the mycelium, develop concentric rings and often coalesce finally. The older stems and also the flowers and fruit are not attacked by the fungus.

A cross-section of the stem shows that the vascular bundles are coloured brown. On the under side of the leaf-spots, on the light green portion, there arises a network of delicate brownish-grey hyphæ which are short and unbranched. At the tip of each hypha an ovate sporangium is abstricted which either germinates by zoospores (5–15 in number) or by a single germ-tube. Another form of spore—the chlamydospore—has been observed on inoculated fruits. These spores are globose, yellow, and have thick, smooth walls; they may germinate at once or after some time, and they can infect the soil for several months.

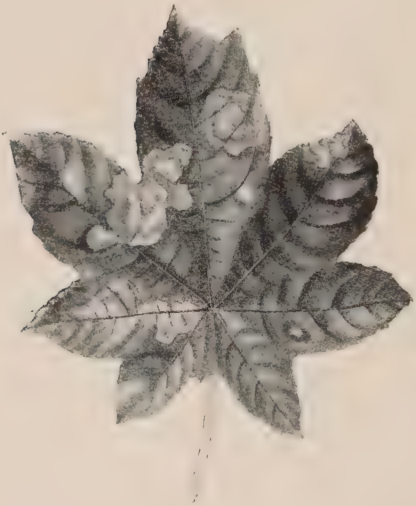

FIG. 64.—*Phytophthora parasitica.* Diseased leaf of *Ricinus communis.* (After E. J. Butler.)

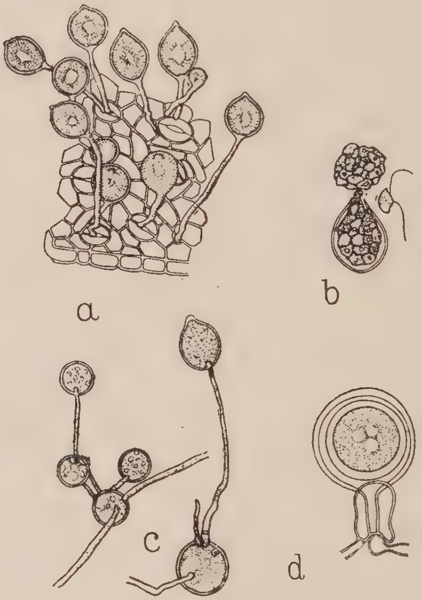

FIG. 65.—*Phytophthora parasitica* on *Ricinus communis.*

a, leaf surface with sporangia; *b*, sporangia germinating; *c*, chlamydospores; *d*, oospore with portion of antheridium. (After E. J. Butler.)

The method of over-wintering has not been clearly proved; oospores have not been found to occur naturally but only under artificial conditions. It was observed that silkworms do not feed on the diseased leaves.

This fungus is regarded as the most destructive pest met with in growing the castor-oil plant; it has also appeared on *Sesamum indicum* and has been transmitted, by artificial inoculation, to young plants of *Solanum tuberosum*, *S. Lycopersicum*, *S. Melongena*, *Fagopyrum*, *Œnothera*, *Schizanthus*, *Clarkia*, *Gilia*, and *Salpiglossus*, but not to *Phaseolus*, *Nicotiana*, *Lepidium*, *Jasminum*, *Cereus*, *Opuntia*, *Colocasia*, *Cleome*, and *Panax*.

The disease was noticed for the first time in 1909 in Pusa (India), and since then it has appeared each year from June to September (Dastur, 1913). In England it has caused serious damage to the tomato crop (Bewley, 1921), and it has also appeared on tomatoes in Holland (van Poeteren, 1925).

To control the disease it is recommended that the castor beans which are to be used as seed should come from a crop which has been inspected previously and found to be healthy.

In the United States (Columbia, Maryland, Virginia) a similar fungus was observed on rhubarb in 1917 and has been classified as *Phytophthora parasitica* var. *Rhei* (Godfrey, 1923).

Phytophthora Thalictri Wils. and Dav. on *Thalictrum purpuraceum* and *Th. polygonum* in the United States (Clinton, 1909) and **Ph. (Blepharospora) terrestris** (Sherb.) Peyr. on the roots of *Lupinus albus* (Peyronel, 1920) also come into this section.

Downy Mildew of Beet

Peronospora Schachtii Kühn

The first symptoms of the disease appear in spring on the leaves of seed beets, and the infection spreads to neighbouring newly-planted fields. The diseased leaves are of a pale green colour, curled, and with the margins curved outwards. In the later stages, the leaves are swollen on both sides, but especially on the under side, and covered with an ashy-grey or yellowish felt composed of branched hyphæ bearing at their tips ovoid conidia which serve to spread the disease. The infected leaves die prematurely. In old, withered leaves there are sometimes produced thick-walled oospores by means of which the fungus hibernates. It is thought that the fungus lives from year to year as a sterile mycelium in the crown of seed beets which are being kept over the winter.

The intensity of attack varies from year to year; if the season is wet considerable damage may be done to a crop, but if dry the losses from the disease are small. Severely infected crops may be reduced to the extent of 20–30 per cent. of the weight of the roots. The disease also checks the formation of seed where the crop is being grown for this purpose.

Sometimes in the height of summer—middle or end of July—individual plants in a field of sugar-beet have become diseased (in Sweden, 1921 and 1922), the leaves in the centre of the head being small, thickened and much shrunken. No disease was found on surrounding plants during the growing season, and it must be assumed that infection came through the seed.

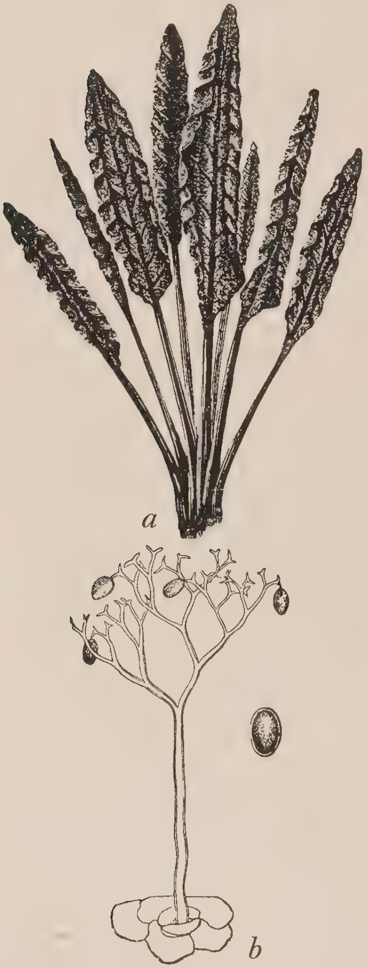

Fig. 66.—Downy Mildew of Beet. *a*, young leaves with backward rolled margins; *b*, dichotomously-branched hypha with conidia. (After E. Rostrup.)

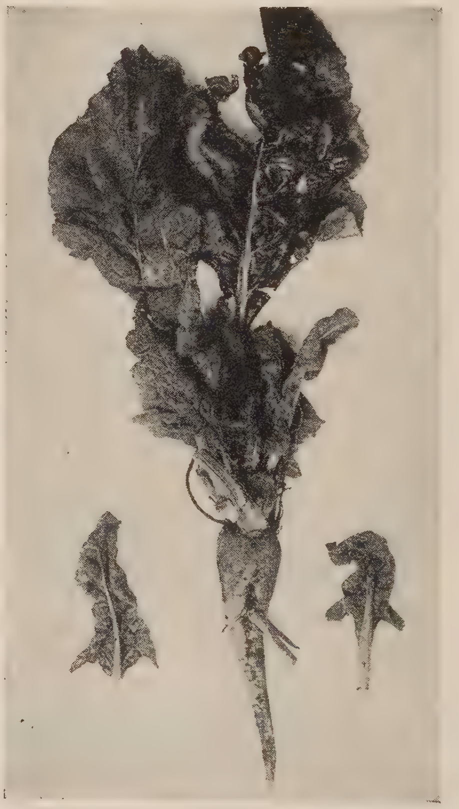

Fig. 67.—Beet Plant attacked by Downy Mildew in Summer. Taken from an otherwise healthy crop. (Orig.)

Preventive Measures:

(1) Roots to be grown for seed must not be taken from an infected

field. (2) Seed beets should be placed as far away as possible from the fields on which roots are being grown. (3) All suspicious roots should be rejected when planting out in spring for seed production.

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Downy Mildew of Lucerne

Peronospora Trifoliorum De By.

The fungus which is the cause of the disease attacks not only the leaf and leaf-stalk but also the stem of the plant. Chlorotic patches of varying size are produced, often in the shape of broad, grey bands across the leaf. The leaves become bent and shrunken, and in severe cases the whole plant is misshapen, only short, swollen stems and rudiments of leaves, which are covered with a grey coating, being formed. The plants do not flower and the field can be recognized from a distance by its variegated foliage. The patches are found mainly on the under side of the leaf and are covered with a grey or pale lilac growth, the branched hyphæ of which produce spherical conidia on the outside branchlets. In the withered parts of the plant, especially in the dead stipules, thick-walled oospores are found, and by means of them the disease is carried on from year to year.

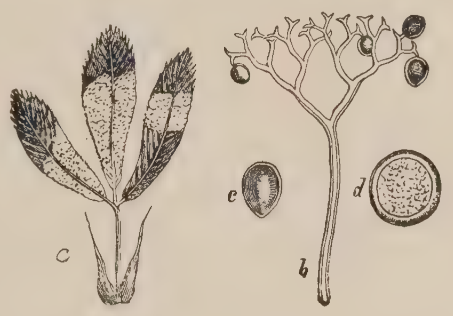

FIG. 68.—Downy Mildew of Lucerne. *a*, leaf with light spots of fungus; *b*, hypha with conidia; *c*, conidium; *d*, oospore. (After E. Rostrup.)

The disease is also found in the first year of newly laid-down fields of lucerne, more particularly—according to European experience—where the seed has been of American origin. The conclusion drawn from observations made in 1884 and 1885 in Denmark and in Sweden in 1889 is that the disease can be transmitted by infected seed.

It has not yet been decided whether the forms of fungi which attack *Medicago falcata* and *M. lupulina* as well as those on different species of the genera *Trifolium*, *Lotus*, *Melilotus*, *Astragalus*, *Ononis*, and *Coronilla* are really identical with the one described above.

This fungus was described in 1863 and recorded as being destructive to lucerne in England in 1884. It has been known in Denmark since 1885, Sweden 1889, the United States 1907. In the last-named country it has spread to such an extent that in 1913 it was found generally from New York to California. It appeared in Australia (Victoria) in 1911.

Preventive Measures :

(1) Great care must be taken to procure uninfected seed, and the use of American seed should be avoided. (2) A lucerne field which shows signs of disease in the first year should be ploughed up.

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Peronospora Viciæ (Berk.) de By. appears on numerous species of *Vicia*, *Pisum*, *Lathyrus*. *Ervum*. *Orobus*. etc. The fungus produces greyish-violet patches (which sometimes coalesce) on the under side of the leaf. These patches consist of much-branched conidiophores which abstrict conidia at the tips of the branches. Oospores are found inside the infected leaves. The disease may, in the case of several varieties of *Vicia* and *Lathyrus*, considerably reduce the crop.

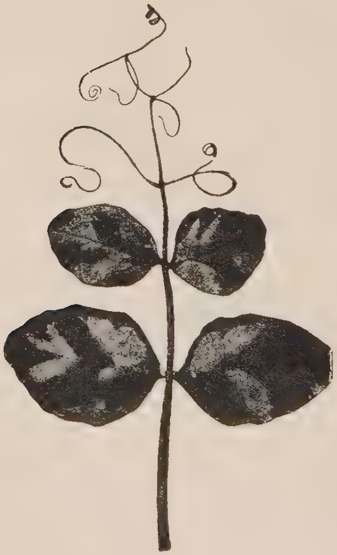

Fig. 69.—Downy Mildew of Pea. (After E. Rostrup.)

Peronospora parasitica (Pers.) de By. This fungus produces a snow-white growth on the green parts of young crucifers. The conidiophores are repeatedly branched and an elliptical spore is produced at the tip of each branch. On the stems and flower stalks gall-like swellings often occur, and in them oospores are found. The fungus attacks many brassicas, *e.g.* cauliflower, brussels sprouts, kohl-rabi, turnips, colza, and rape, as well as other garden plants such as radishes and mustard, and certain flowers. *e.g.* species of *Matthiola*, *Cheiranthus*. *Hesperis*. Numerous uncultivated species of the genera *Capsella*, *Thlaspi*, *Draba*, *Lepidium*, *Cardamine*. *Sinapis*, *Erysimum*. *Alliaria*. *Dentaria*, etc., are also attacked. According to recent investigations, the fungi found on the different species of domestic plants are distinguished from one another not only by biological characters but also by morphological differences such as shape and size of conidiophores, size of conidia, markings, and diameter of oospores, etc. No less than fifty-two different forms named after their respective host-plants (e.g. *Peronospora Brassicæ*, *P. Alliariæ*, etc.) have been distinguished, the majority when cultivated artificially, have shown themselves to be sharply fixed to definite species of host-plants (Gäumann, 1918). This fungus is known throughout the world and not rarely it occurs together with white rust (*Cystopus candidus* (Pers.) Lév.).

Other species of Peronospora which are injurious to agricultural plants are the following: **Peronospora obovata** Bon. on *Spergula arvensis* and *Sp. pentandra;* the stem and leaves are covered with a grey growth and the plants wither early. **P. arborescens** (Berk.) de By. found on seedlings and sometimes on young flower stalks of *Papaver somniferum*, *P. Rhœas*, etc.; the infected flower stalks often show gall-like thickenings, and become twisted. **P. cannabina** Otth. on *Cannabis sativa;* yellow spots covered with a dark grey growth on the under side, particularly in the angles of the veins. **P. Maydis** Rac. on *Zea Mays;* the leaves of young maize plants become greenish-white; the stem soon decays and the whole plant collapses. **Plasmopara nivea** on carrots and parsnips; the leaves are covered on the under side with a snow-white growth and soon become flaccid.

Downy Mildew of Spinach

Peronospora Spinaciæ (Grev.) Laub.

A few weeks after the seed is sown, the disease makes its first appearance on the leaves in the form of irregularly-dispersed patches, well separated from one another. The upper side of the patch is yellowish-white and the under side covered with a greyish-blue growth which later on almost completely covers the whole surface of the leaf; the leaf lamina is often buckled and the margin incurved. In severe attacks only the first leaves can be used and the infected plants should be removed at once and fresh seed sown.

The downy covering is formed of closely-placed, delicate, upright dichotomously-branched conidiophores, which produce ellipsoid conidia at their tips.

The fungus does not appear to produce oospores but to be carried on from year to year as a latent plasm in the seed produced by diseased plants. A microscopical examination of the leaf cells—palisade as well as spongy tissue—of a plant grown from infected seed shows turbid masses of protoplasm which are not found in the leaf-cells of a healthy plant. These masses when stained have been seen to consist of a colloidal fluid in which are embedded minute threadlike or granular bodies. The turbid mass is the result of the symbiotic coalescence of the protoplasm of the host-cell with that of the fungus; it is the so-called mycoplasm in the resting stage. There are also certain differences to be observed in the shape of the chloroplasts of a healthy stem and those of a diseased one, the former being less dense and with an uneven surface, *i.e.* they appear to be in a state of dissolution. At a later stage of development, the chloroplasts dissolve and their contents pass into the plasm. A similar change also takes place in the case of the nuclei of the cells. In this reinforced plasm-mass, which completely or partially fills the host-cell, there arise several (3–10) nucleoli of varying sizes (mycoplasm in the nucleolar stage).

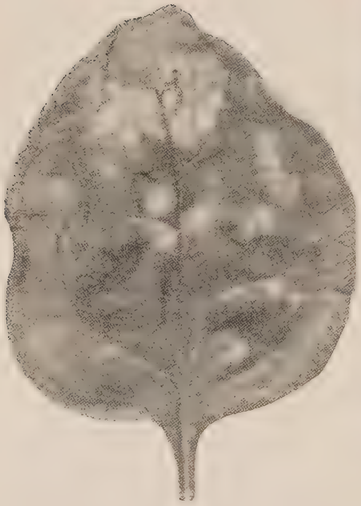

Fig. 70.—Downy Mildew of Spinach. (Orig.)

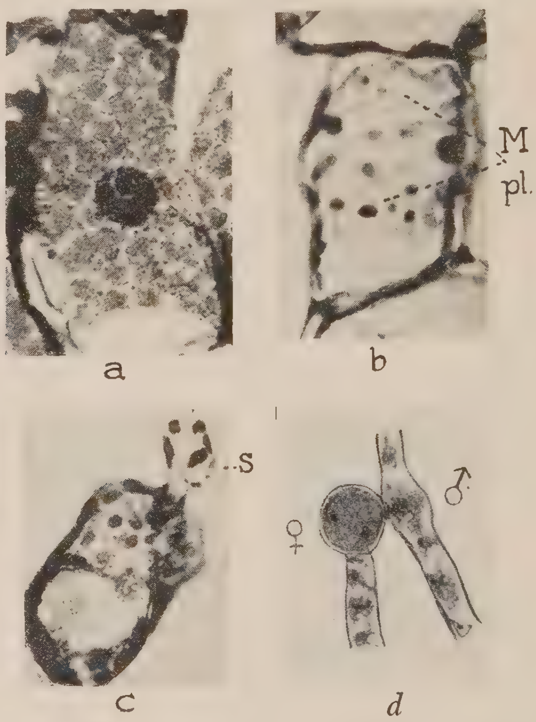

Fig. 71.—Downy Mildew of Spinach.

a, mycoplasm in resting stage; *b*, mycoplasm in ripe stage, with nucleoli; *c*, extrusion of plasm; *d*, fertilisation of oogonium by antheridium. (Orig.)

With the dissolution of the chloroplasts and the appearance of the nucleolar plasm the maturing stage of the mycoplasm has begun and the intracellular fungus is ready to enter upon an independent existence in the intercellular spaces.

From a large intracellular accumulation of material the fungus passes, probably aided by passages in the cell wall, as a wide tube into the intercellular space. Once the fungus-tube has passed out, it develops further and assumes the character of a true mycelial hypha. Certain of the mycelial hyphæ develop into male organs (antheridia) and others into female (oogonia). By the coalescence of the contents of these organs, oospores are formed; these are true summer spores and can germinate at once. The germ-tube which passes out through a stoma develops into a dichotomously-branched mycelium from which conidia are produced. These primary conidia function as zoosporangia, that is, their contents divide into a number of zoospores which germinate at once when freed.

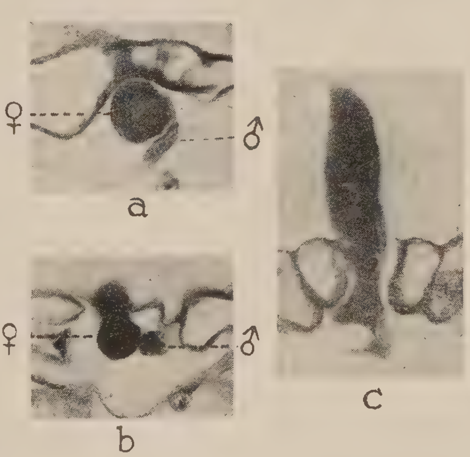

FIG. 72.—Downy Mildew of Spinach.

a, finished oospore with remnant of antheridium adhering to it; *b*, germinating oospore; *c*, germinated oospore. (Orig.)

Various hypotheses have been advanced to explain the occurrence of the fungus on new spinach crops. The absence of oospores in the plants has led to the oospores of the closely-related fungus on *Chenopodium album*, etc., being regarded as the cause of the primary outbreaks. This fungus was, for a long time, thought to be identical with the one on spinach. It has also been suggested that the reappearances of the disease is due to hyphæ which are produced in the spinach beds in autumn and carry the fungus over the winter. Neither of these explanations has, however, been able to withstand criticism.

The disease was first reported from Edinburgh (Scotland) in 1824, and the organism named *Botrytis effusa*. Later investigators identified it with *Peronospora effusa*, which is found on numerous other members of the Chenopodiaceæ family (*Chenopodium*, *Blitum*, *Atriplex*); and finally in 1906 the fungus was classed as an independent species under the name *Peronospora Spinaciæ* (Grev.) Laub. During the past forty years the disease has been observed in numerous places in Europe and North America.

Control Measures :

To ensure a healthy crop only such seed should be used as is known to have come from healthy plants.

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A fungus causing Downy Mildew on buckwheat (*Fagopyrum esculentum*) has been observed in France (Ducomet, 1911) and is closely related to the above fungus on spinach.

Downy Mildew of Onion

Peronospora Schleidenii Ung.

This disease occurs sporadically in summer on the leaves of the onion in the form of a whitish growth with scattered violet-brown dots ; on seed onions the disease is sometimes seen in April. In Denmark it has been suggested that there is a primary outbreak arising from seed onions, and a secondary outbreak which spreads from plant to plant (Rostrup, 1902). The growth on the leaves consists of numerous, branched hyphæ which abstrict obovate conidia. The conidia germinate by a tube, and in damp, warm weather the infection spreads rapidly over a large area in a single night. The upper portion of the leaves of young plants often dies quickly. Dry weather checks the spread of the disease, and the infected leaves wither. In August the whole of the leaves may be bent downwards and dead. If the attack is severe the leaves and the bulb become rotten. Oospores are found in the dead tissues, and it is believed that they retain their viability for 2–3 years. The fungus also over-winters as a non-septate mycelium in the infected parts of the onion.

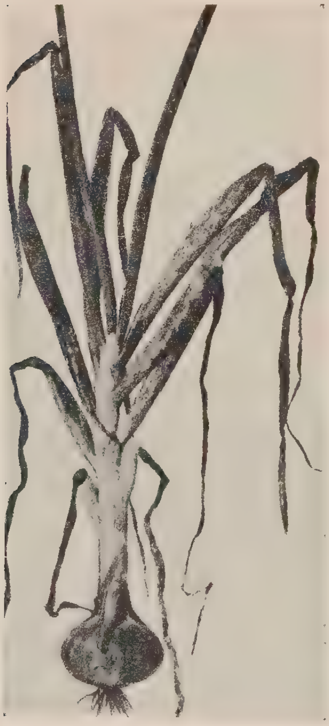

Fig. 73.—Downy Mildew of Onion. (After J. C. Walker.)

Primarily the fungus attacks *Allium Cepa* and *A. fistulosum*, but it is also found on other species of onion and at times even on species of *Lilium* and *Amaryllis*.

This disease was first observed in England in 1841, and since the middle of the last century it has been common in most European countries where onions are grown. In 1872 it appeared in the United States ; severe losses through the disease have been reported from Sweden, 1882–87, and again in 1905–11 ; Italy, 1890–92 ; Germany, 1894 and 1898 ; Denmark, 1902, 1904, and 1922 ; Norway, 1922 and 1923. Since 1904 the disease has been widespread in North America—from New England to Wisconsin.

Control Measures :

(1) The ground on which onions are grown should get the morning sun and a free circulation of air. (2) If the primary outbreak is limited in extent and a cold, dry period supervenes all the infected plants should be collected and burnt. (3) All fragments from diseased plants should also be burnt. (4) Onions should not be grown on the same soil year after year. (5) An infected crop should be sprayed with Bordeaux mixture in the early morning whilst the plants are still wet with dew or immediately after rain.

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Downy Mildew of Rose

Peronospora sparsa Berk.

Numerous scattered spots of irregular shape appear on the leaves and flowering shoots, especially in the case of roses grown in greenhouses. The spots on the upper side of the leaf are pale red to purplish-red at first, finally becoming brown, whilst those on the under surface have a whitish-grey coating. On the stem there are long, dark brown patches covered with a felt of fungus. At the apex of the annual shoots the leaves are misshapen and more or less crumpled or rolled together. The flower buds are checked in their development, the diseased leaves fall prematurely, as do also the shrivelled flower buds, the stems and branches decay and die. The growth on the under side of the leaf-spots consists of numerous delicate conidiophores which branch frequently, and at the tips of the last branchlets, which are curved to a hook shape, ovate or globoid conidia are abstricted. In a moist environment, the disease spreads very rapidly by means of the conidia, which germinate easily. Yellow, thick-walled oospores are found in the brown, dead leaves, and in the cortex of the stem of infected shoots; the oospores carry the disease over the winter.

This disease, which probably came to Europe from India, was observed for the first time in 1862 in a cold glasshouse in England; about fifteen years later it caused much damage in Berlin glasshouses. In 1884 a grower near Odensee in Denmark, lost more than two thousand rose trees. From this period down to the present time, almost all European countries furnish examples of the devastating effects of the disease and the rapidity with which it spreads. The introduction of infected rose stocks has been the cause of many serious epidemics of the disease.

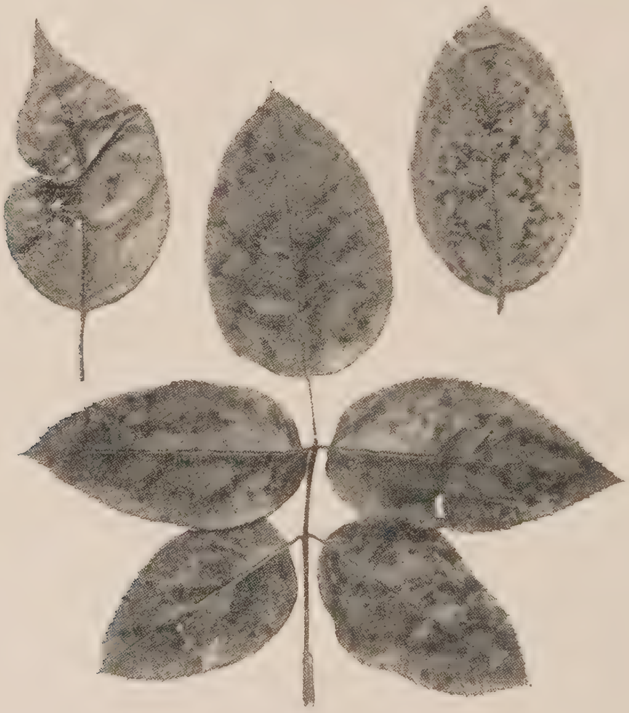

Fig. 74.—Downy Mildew of the Rose. (Orig.)

Preventive and Control Measures:

(1) Every care should be taken to ensure that imported rose trees or

rose stocks come from a disease-free nursery. (2) If disease appears, the glasshouse should be kept dry and well ventilated, or the plants should be removed to an open hot-bed out of doors. (3) Carefully collect and burn all infected leaves, flowers, and buds. (4) Disinfect the soil with 1 per cent. formalin solution in late autumn and again with 0·5 per cent. solution two weeks before planting.

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Peronospora Cytisi Rostr. attacks *Cytisus Laburnum* and *C. alpinus*, sometimes also other species of *Cytisus*. The fungus attacks the seedlings preferably, and the diseased leaves become marked with brown spots and soon wither, the seedling dying within a few days. On the under side of the leaf-spots, an ashen-grey coating composed of very delicate conidiophores is formed and from the tip of each conidiophore an elliptical conidium is abstricted. Numerous oospores are contained within the tissues of the spots.

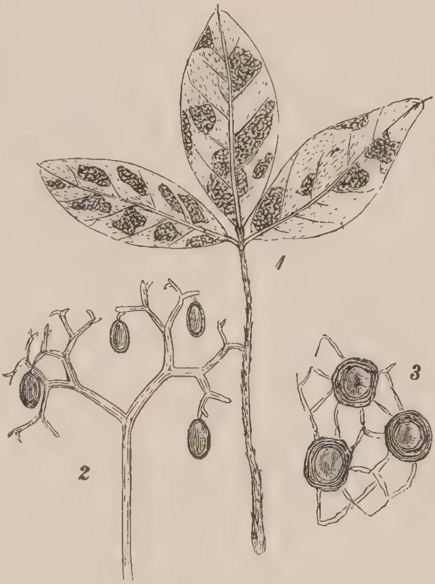

Fig. 75.—*Peronospora Cytisi*.
1, diseased leaf; 2, conidiophore with conidia; 3, oospores. (After E. Rostrup.)

The disease was found in a nursery in Denmark (Roskilde) in 1888 and 1890, and in Germany in 1888 and 1891.

On older plants, the younger leaves at the upper end of the twig are especially liable to attack. Numerous light brown spots on the lamina, which is also bent abnormally, are characteristic. The life of the shrub is not endangered (Rostrup, 1892; Kirchner, 1892; Magnus, 1892).

As a preventive measure it is recommended to spray the diseased plants at once with Bordeaux mixture and not to use the infected ground for laburnum in the next year.

The following forms also come into this section :—

Peronospora Dipsaci Tul on *Dipsacus fullonum*, the scales on the head of the teasel and also the leaves have a pale downy mildew covering. **P. calotheca** de By. on *Asperula odorata* and species of *Galium*. **P. Violæ** de By. on species of *Viola*. **P. Oerteliana** Kuhn on *Primula officinalis*. **P. Alsinearum** Casp. on species of *Cerastium*, *Alsine*, *Arenaria*, and *Stellaria*. **P. Hyoscyami** de By. on *Hyoscyamus niger*, yellowish, discoloured spots on the leaves of older seedlings ; the plants in some cases are killed (Peters, 1912). **P. Arabidis** Gäum. on species of *Arabis* (Gäumann, 1918). **P. pulveracea** Fuck. on species of *Helleborus* (Klebahn, 1925).

Downy Mildew of Vine

Plasmopara viticola (Berk. & Curt.) Berl. & de Toni.

This disease appears in Mid-Europe in June–July, and in southern climates in May. It manifests itself by the production of irregularly shaped spots on the leaves of the vines, especially on certain American varieties such as *Vitis æstivalis*, *V. Labrusca*, *V. vulpina*, and *V. cordifolia*, and latterly also on the native European variety, *V. vinifera*. The upper surface of these leaf-spots, which have, on account of their appearance, been termed " oil spots," is yellow at first and later turns red, whilst the under side is covered with a white, downy growth. As the disease advances the leaf curls up, withers and falls. The white downy coating, which may also be present on very young shoots, pedicels, flowers, and newly-forming fruit, consists of much-branched conidiophores, several of which emerge from a stoma. The conidiophores, at the tips of which conidia are formed, usually push through the stomata during the night, particularly if there is a change from cool to warm weather.

The conidia when ripe, which is usually 1–2 days after they have been formed, germinate in a few hours if placed in a drop of water. In the process of germination the contents of the conidium generally divides into 6–8 smaller portions which pass out through a hole in the apex of the conidium ; thus the conidia behave like sporangia. The freed zoospores, which are provided with two flagellæ, round off after about half an hour's movement, come to rest, and then each zoospore puts out a single germ-tube. In rare cases, when the temperature is below 20° C., the conidia germinate differently, the whole of the protoplasm passes out through an opening at the apex of the conidium, rounds off, and then sends out a germ-tube.

Continued moist and warm conditions greatly favour the spread of the disease, and its appearance on the leaves seems to be especially dependent upon two fairly heavy falls of rain. After the first fall, zoospores are produced and infection follows, whilst after the second fall of rain conidia are produced and fresh infection results. Strong winds before or during a thunderstorm are very favourable to the dissemination of the conidia.

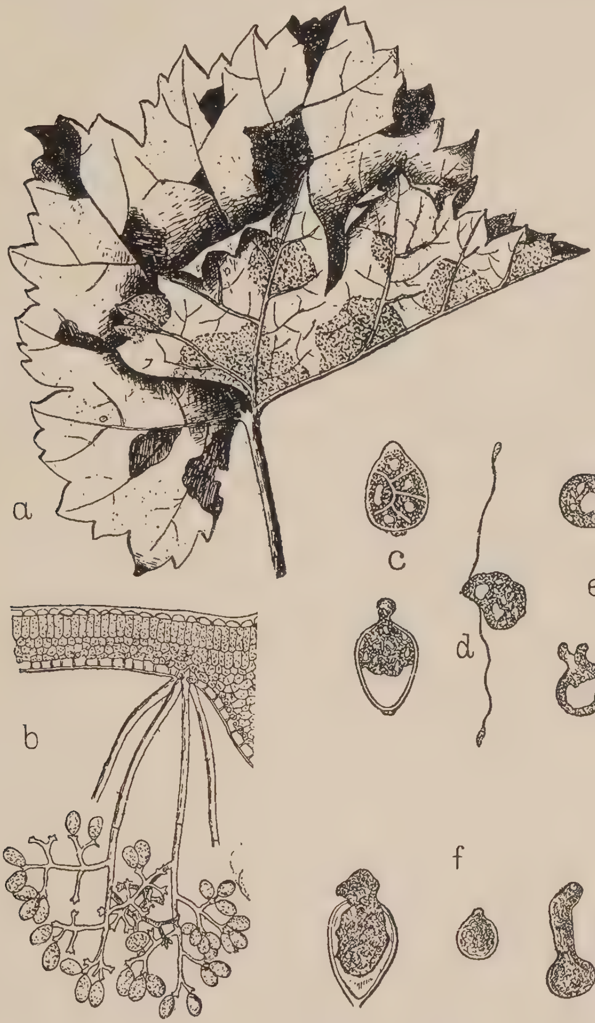

FIG. 76.—Downy Mildew of Vine.

a, diseased leaf; *b*, cross-section of leaf with bundle of conidiophores; *c*, zoosporangia; *d*, zoospore; *e*, germinating zoospores; *f*, zoospore germinating abnormally. (*a* and *b*, after T. Ferraris; *c–f*, after P. Viala.)

Infection takes place principally on the lower surface of the leaf, where stomata are present in much larger numbers (about 2 million on each 10 cm. of vine leaf) than on the upper surface, where the number is only some 2–4 thousand per 10 cm. (Ruhland and von Faber, 1909; Müller-Thurgau, 1911), and partly through the pores on the margins of the leaf (Kroemer, 1921). The germ-tubes of the zoospores enter by the stomatal openings and the period of incubation after such an infection varies from 7–14 days, according to the weather and the time of the year; as summer advances the period decreases.

Continued dry weather, in which any rain that may fall quickly dries up, can cause the spread of the disease to cease, in fact infected leaves may in such circumstances recover.

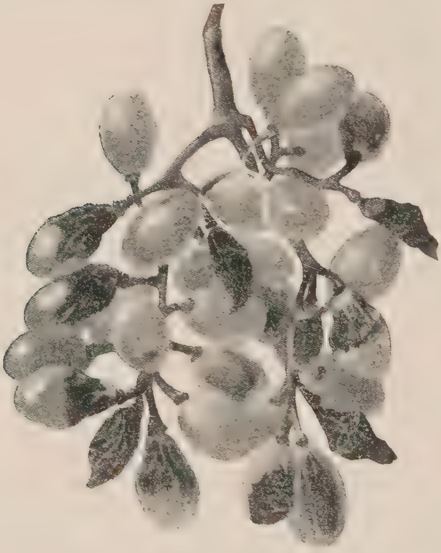

Fig. 77. -Downy Mildew of Vine. Diseased grapes. (After G. Linhart and G. Mezey.)

When the grapes are attacked at a later stage a few of the berries may sometimes develop normally; most of them, however, become brown and shrunken. Where infection of the grapes takes place at an early stage, growth ceases and the berries are small, shrivelled and dry.

In autumn, after the leaves have changed to a bleached, whitish-grey colour, another type of conidia formation is found on the lower surface of the leaf. A tuft of short-stalked hyphæ appears at one of the stomatal openings, and from the tip of each hypha a conidium is abstricted. These conidia are larger than those produced by the conidiophores and are termed macroconidia; they germinate by a single germ-tube in the same way as the true conidia. The part that they play in the life-history of the fungus is not known, as apparently inoculation experiments with them have not yet been carried out.

Finally, in late autumn, the real resting spores of the fungus—the oospores—are to be found in the palisade cells and spongy tissue of the leaves. These spores, which are nearly spherical in shape, have a thick, hyaline inner wall and a very thin, transparent outer wall. They over-winter with decaying leaves on the surface of the soil and infect the young leaves in the following year. These oospores are also met with in dead branches and fruit, sometimes even in the older parts of the stem.

The manner in which oospores germinate, whether by a germ-tube or by zoospores, is not certain (Delacroix and Maublanc, 1909), nor is it clear what part they play in the reappearance of the disease in the following year (Duggar, 1909). As it has been found that the mycelium of the fungus can withstand a temperature several degrees below freezing-point, it is possible that an over-wintering mycelium has also to be reckoned with (Ravaz, 1913).

The disease appears to be endemic in North America, where it was met with for the first time in 1834, but the description of it was not given until 1851. Originally the disease was limited to the Mississippi Valley, but it soon spread northwards and eastwards, and it is now found in vineyards throughout the whole of the North-American continent.

The importation of American vines (which were considered to be less susceptible to the Phylloxera disease) was undoubtedly the cause of the outbreaks of Downy Mildew disease in Europe in 1878, a consequence already predicted in 1873 by the French investigator Maxime Cornu. The first outbreak was in the south-west of France, and two years later (1880) the vineyards in mid- and northern France, Alsace, Würtemberg, Silesia, Italy, Algiers, Portugal, Tyrol, Hungary, Greece, etc., suffered more or less severe losses. The fungus has been known in Australia since 1916–17 (Victoria), and 1920–21, Queensland (Osborne and Samuel, 1922).

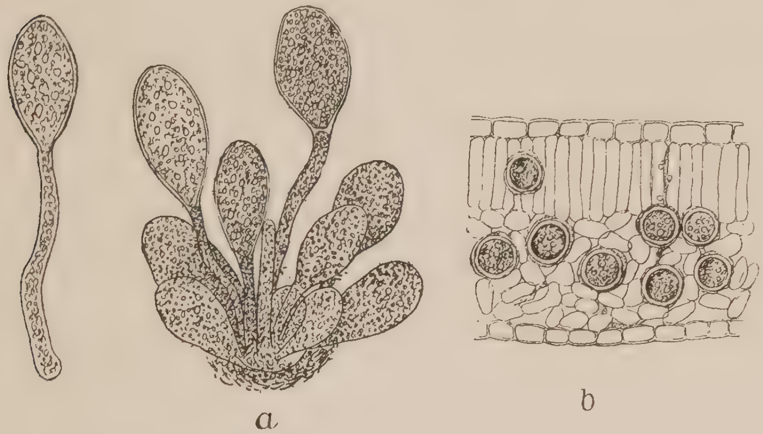

FIG. 78.—Downy Mildew of Vine.
a, conidia ; *b*, oospores. (*a*, after P. Viala ; *b*, after E. Prillieux.)

The losses which the disease has caused have been most serious in some years. Thus in 1906 it is estimated that for the whole of the German vineyards the loss was equivalent to more than 1½ million pounds sterling, as compared with the average of the years 1902–05. In France, in 1915, it was calculated that 70 per cent. of the crop was lost, due very largely to the fact that at that time labour was not available to undertake the necessary spraying measures (Caloret, 1923).

The best results in the control of the disease have, on the whole, been obtained by the use of Bordeaux mixture (copper sulphate and lime) or Burgundy mixture (copper sulphate and soda). The spray must be applied to the under surface of the leaf by means of a special nozzle, and three sprayings during the growing period are usually regarded as sufficient.

The first spraying is of the greatest importance and must be made as soon as the primary " oil spots " appear. To ensure that this stage shall not be missed it is the custom in many vine-growing districts to award prizes to those who first notify it. In addition, much investigation has been made into the connection between the outbreak of the disease and weather conditions. It has been found, for example, that the primary outbreaks of disease (set out graphically as " Isophanes ") regularly run parallel with the heavy early rains (set out graphically as " Isohyetes "), and by making use of the " Isohyetes " the " Isophanes " can be determined with considerable accuracy (Savoly, 1912).

It is known that the fungus needs at least two heavy falls of rain for its development; with the first fall it penetrates into the green tissues and remains there for a definite time, in May for 15–18 days and in late July 5–6 days (incubation period) in the case of Hungary. Shortly afterwards, if the moisture conditions are favourable, the white growth of conidia appears (de Istvánffi and Pálinkás, 1912). If, therefore, the date of the primary attack be known and to it be added the appropriate incubation period, then the time at which the outbreak of the disease is to be expected can be judged and steps taken to prevent it by early spraying.

Working on the above lines it has been found possible to prepare charts by the use of which the vine grower can calculate when and how often he must spray his vineyard (K. Müller, 1918). In Italy a similar system is also used, and advice based on meteorological observations is issued to vine growers through the daily newspapers during the months of April and May (Voglino, 1922, 1925).

In France definite periods for spraying the vineyards have been recommended; thus north of the line Bordeaux–Briançon (*a*) twice, June 1–8; (*b*) twice, June 15–25; (*c*) once, about July 10; whilst south of this line (*a*) twice, May 15–23; (*b*) twice, June 1–9; (*c*) once, about July 10. Similar recommendations have also been drawn up for Algeria and Tunis. For the first two sprayings the Bordeaux mixture should be made from 2½–3 kg. copper sulphate, 2½–3 kg. lime, and 100 litres water, whilst for the last spraying the proportions should be 1 : 2 : 100 (Caloret, 1923).

Preventive and Control Measures :

(1) Remove and destroy all diseased leaves and tendrils. (2) If all diseased parts of the plant are not completely removed in winter, dig over the soil near the stock of the vine and sprinkle powdered copper sulphate over the ground. (3) When the primary outbreak of the disease is to be expected (according to the meteorological data) spray with 1–2 per cent. Bordeaux mixture or Burgundy mixture. A second spraying should follow a few weeks later (if, in the meantime, heavy showers have fallen), and a third spraying a few weeks later again. (4) Varieties selected for planting should be such as local experience has shown to be resistant in a high degree to the disease.

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Latterly the fungus has been found on *Ampelopsis Veitchii*. Scattered angular spots, brown to red in colour, appear on the young leaves at the beginning of September ; on the under side these spots have the characteristic downy growth. During the period 1904–21, and especially in the years 1904 and 1913, the disease was found in Geisenheim (Muth, 1925), and in the abnormally wet year of 1924 in the Rhine Province (Lüstner, 1924).

Downy Mildew of Cucumber

Plasmopara Cubensis (Berk. & Curt.)

This disease appears about the middle of June or the beginning of July in the form of angular spots on the leaves of cucumbers and melons. The spots are at first of a yellowish-green colour, later becoming dirty yellow, and they are sharply limited by the leaf veins. On the under side of the spots, a loosely woven violet-coloured growth is seen. The growth is an aerial mycelium the main branches of which, usually 1 or 2, more rarely 3-5, grow out through a stomated opening. The mycelial hyphæ are somewhat swollen at the base and vary in length according to the humidity of the air; they branch dichotomously at the tip. The ultimate branches each bear a spore with a brownish-violet membrane and an apical papilla. When the spores are placed in water they germinate as sporangia with zoospores, but sometimes also as conidia by a single colourless thin-walled tube which is put out at any point on the sides of the spore or at the apex.

The fungus—which is sometimes given the generic names *Pseudoperonospora*, *Pseudoplasmopara*, or *Peronoplasmopara*—requires a very high temperature. The infected leaves either rot or dry up according to the humidity of the atmosphere, and as a consequence the plant is seriously checked and the formation of fruit much diminished.

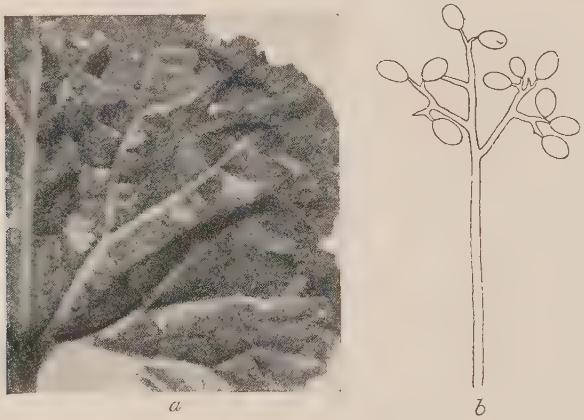

FIG. 79.—Downy Mildew of Cucumber.
a, diseased leaf; *b*, conidiophore. (*a*, after C. P. Clinton; *b*, after E. J. Butler.)

The fungus attacks numerous species of Cucurbitaceæ such as *Cucumis sativus*, *C. Melo*, *C. Anguria*, *Citrullus vulgaris*, *Cucurbita pepo*, *C. moschata*, *Sicyos angulatus*, etc., it restricts itself to plants in this family. Cucumbers are more susceptible on the whole than melons, and the latter more than pumpkins. It has also been observed that individual sorts of cucumbers possess a different degree of susceptibility to the disease.

The fungus was first observed in Cuba in 1868; in 1876 it appeared in Russia; in 1889 in Japan, and very seriously in the United States. It has been known in England since 1902, and since 1903 in Austria, Hungary, and Italy. Owing to the losses caused by this disease the growing of cucumbers and melons has been abandoned in many places. Infection is probably spread through the seed, but how this occurs is not known.

Preventive and Control Measures :

(1) Seed from healthy plants should be used. (2) Glasshouses in which the disease has appeared should be washed or sprayed with 0·8 per cent. formalin solution; all implements should be thoroughly disinfected. (3) Spray each 14 days with 1 per cent. Bordeaux mixture from the time the plants begin to develop. (4) Cucumbers grown out of doors should be sprayed also with Bordeaux mixture every fortnight.

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Downy Mildew of Hop

Pseudoperonospora Humuli Miy. & Tak.

The disease can be recognised in early summer by the appearance on the leaves of reddish-brown, angular spots, which occur mainly alongside the midrib. The under surface of these spots is pale in colour and on it is found a blackish-grey fungus growth which is sometimes tinged with violet. This growth consists of long, dichotomously-branched conidiophores, and at the tips of the branches oval conidia are abstricted. The conidia on germination produce zoospores which spread the disease so rapidly that in damp weather the leaf may be covered with fresh spots within a week. At a later date the fungus appears on the hop cones, where it causes the bracteoles to turn brown whilst the bracts remain green. The cones thus assume a reddish-brown, checked appearance, and if the attack is severe they may be ruined. The dark-coloured groups of conidiophores which arise on the cones are scattered over the under surface of the bracteoles.

Towards the end of the growing period of the hop, thick-walled oospores, which serve for the over-wintering of the fungus, are found within the tissue of the leaf. The germination of these oospores has recently been observed, and it may be assumed that when the plant decays in the autumn the oospores find their way into the soil and germinate in the following spring or early summer.

This fungus was first observed in Japan in 1905; in 1909 it was found in America, and in 1920 in Europe. The first appearance of the fungus is recorded as occurring about June 15, 1905, in the experimental plot of the Hokkaido Agricultural Experiment Station in Sapporo (Japan). and in the hop-field belonging to the Sapporo Brewery Company. In the same year the fungus was found on the native wild hop, *Humulus Lupulus* L. var. *cordifolius* Maxim. in other parts of Japan. The lower leaves were first attacked and afterwards the cones, which were rendered worthless. In 1916 the fungus was found on *Humulus japonicus*. The first appearance in North America was in Wisconsin in 1909, and it has been reported in other places in 1915 and 1920.

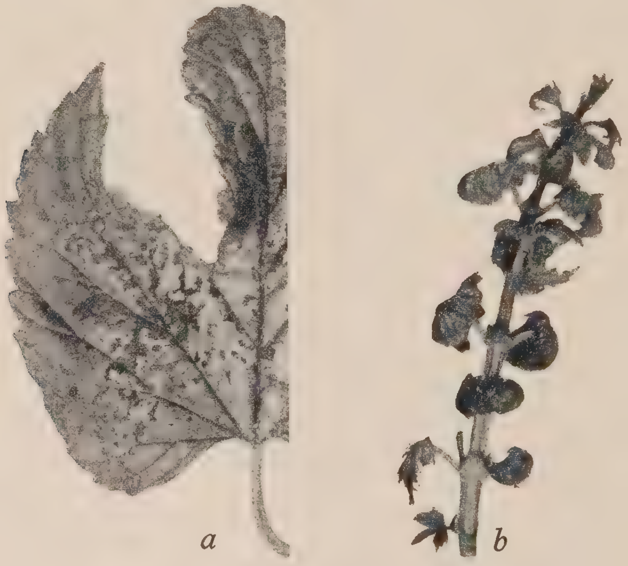

Fig. 80.—Downy Mildew of Hop.
a, portion of infected leaf; *b*, deformed shoot ("spike"). (After E. S. Salmon and W. M. Ware.)

In Europe the fungus was first found, in 1920, in the experimental hop-garden and nursery at Wye College, Kent, on seedlings raised from seed of the Italian wild hop obtained from Italy in 1913. In October, 1920, the leaves of some of these plants showed angular spots with a small amount of fungus growth. The next season (1921) was very dry and the fungus did not appear. In 1922, which was a very wet season, the fungus reappeared on a number of plants of varied origin, causing damage to the leaves and in a few cases also to the cones.

In 1923 the disease in the Wye College garden assumed a new form. In the spring and summer there arose from the root stock or " hill," in addition to the normal climbing bines, short, thick, spike-like shoots. These shoots, to which the name " spikes " has been given in England (in Germany " Bubiköpfe "), vary in length from a few inches to a foot or more, the leaves are small, curled, rather brittle, and closely crowded together. On the under surface of these leaves, and often also on the stem, the fungus appears in greyish-black angular patches. Similar spikes (terminal spikes) may also arise on the bine after it has grown to a considerable height (12–14 feet) above the ground ; such a bine ceases to climb.

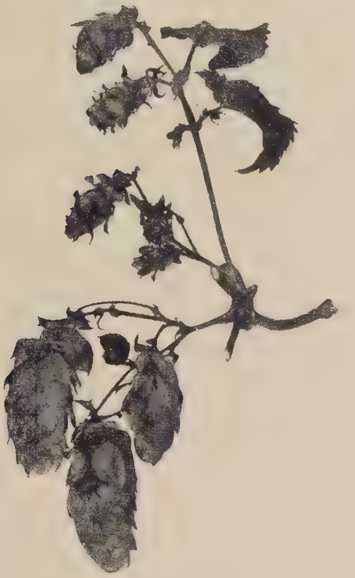

Fig. 81.—Downy Mildew of Hop.
Upper cones diseased, lower cones healthy. (After E. S. Salmon and W. M. Ware.)

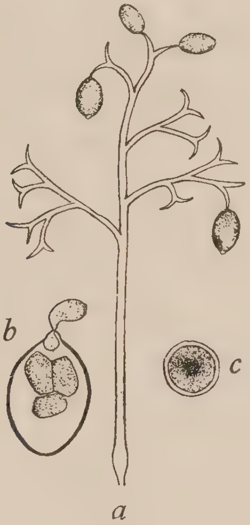

Fig. 82.—Downy Mildew of Hop.
a, conidiophore bearing conidia ; *b*, conidium producing zoospores ; *c*, oospore. (After E. S. Salmon and W. M. Ware.)

In 1924 the disease caused serious damage in many hop gardens, and it was also noted that it occurred on wild hops growing close to nettles (*Urtica dioica* and *U. urens*), which were heavily attacked by a closely allied fungus *Peronospora Urticæ* (Lib.) de By. This observation was responsible for the suggestion that the downy mildew of the nettle and of the hop may be the same species, it having been shown that the former is a member of the genus *Pseudoperonospora* (Salmon and Ware, 1924). Whilst experiments carried out at Wye in 1924 and 1927 have shown that *P. Humuli* is capable of infecting and producing spores on *Urtica urens*, and that spores so produced can infect the hop, the above hypothesis has been abandoned in view of the historical evidence. It is now considered by the above and other investigators that the hop downy mildew is a distinct species which found its way into Europe from Japan, or possibly from North America, about 1920.

There seems to be no doubt that the fungus can be transported from place to place in infected plants or parts of plants. This was shown in 1924 at the East Malling Research Station, Kent, where the disease occurred on plants which had been grown from " sets " obtained from Wye in 1922–23. In the same year the disease was also found in different parts of Kent and Sussex on plants which had originated from East Malling in 1923.

In 1923 the fungus was observed in Yugo-Slavia (Blattny, 1927), in 1924 it was found for the first time in Germany (Würtemberg), in 1925 again in Germany (Bavaria), as well as in Belgium (Capelle St. Ulric, Brabant), France (Grignon and the Côte d'Or), and Russia (Wladikawkaz, Caucasus). In 1926 the disease was reported from a number of other countries—Italy, Austria, Roumania, and Hungary.

It has been found, from the microscopical examinations made at Wye of diseased spiked shoots, that the mycelium of the fungus is present in the inner tissues. The fungus is also found in the rootstock, where it probably exists during the winter and is the cause of the first basal spikes that appear in spring.

Control Measures :

(1) Regular spraying with Bordeaux mixture, of a strength of 1–2 per cent., during the growing season ; as many as ten applications are considered to be necessary in Germany. The spraying must be carefully and thoroughly carried out so as to cover the leaves and hop-cones completely. (2) All diseased parts of the plant, particularly the spiked growths, whether the early " basal " spikes or the later " terminal " or " lateral " spikes on the bine, should be promptly removed and destroyed. (3) New gardens should be placed in well-ventilated situations, not low-lying with a wet soil. Healthy sets should be selected for planting new gardens or for filling gaps in old gardens. (4) Heavy nitrogenous manuring must be balanced with phosphatic and potassic artificial manures. (5) Wild hops (which are commonly infested with the fungus) growing near to a hop-garden should be grubbed up and destroyed.

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Downy Mildew of Lettuce

Bremia Lactucæ Reg.

When this disease attacks a plant, the under surface of the leaf bears large, limited, pale spots, which have a white edging composed of conidiophores and conidia. On the upper surface of the leaf the spots have a bright green or yellow appearance. In spring the spots appear first on the outer leaves near the ground and later spread to the leaves inside the head of the lettuce; finally the whole plant rots and dies.

In the very early stages of the disease, it is possible to save the rest of the head by removing the three or four outer leaves, but such plants are not fit to be sent a distance. When plants grown for seed are attacked at a later stage the crop is sometimes completely ruined.

The dust-like coating on the under side of the spots consists of conidiophores and conidia. The conidia germinate either directly by a germ-tube or by zoospores. It has been suggested that zoospore-formation takes place mainly during the cold period of the year (December to March) and the direct germination in the summer months (Milbrath).

A satisfactory explanation of conidial germination in this species, as in the *Peronosporaceæ* in general, has not been given, but it is not unlikely that the different methods of germination fulfil different biological functions in the life of the fungus, one serving for summer infection and the other for winter infection or for over-wintering.

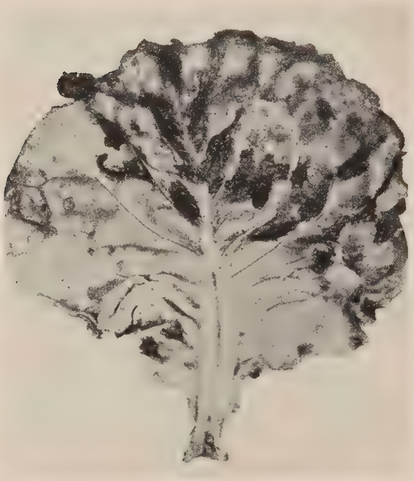

Fig. 83.—Downy Mildew of Lettuce. (After D. G. Milbrath.)

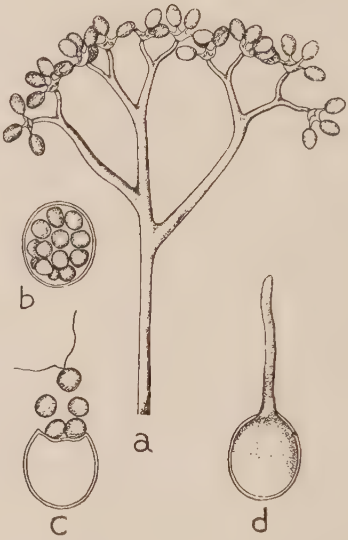

Fig. 84.—Downy Mildew of Lettuce. *a*, conidiophore ; *b*, *c*, *d*, zoosporangium. (After D. G. Milbrath.)

In late autumn, oospores are found in the old decayed parts of the stems of dead plants ; sometimes also in the vessels of the stem. The number of oospores is variable and they serve to carry the fungus over the winter ; they do not germinate until the following spring. In some cases it has been possible to prove the dissemination of the fungus through seed infection (oospore infection), *e.g.* in *Dendrosiris macrophylla* from Juan Fernandez to Bern in 1907, and to Upsala in 1917.

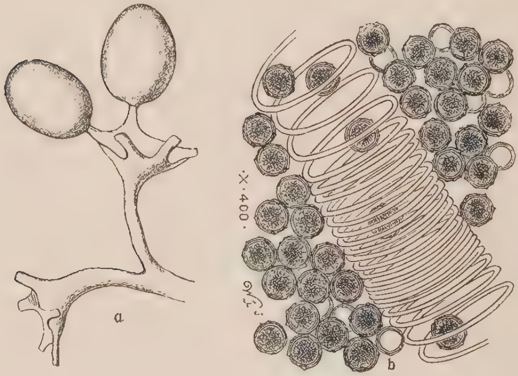

Fig. 85.—Downy Mildew of Lettuce. *a*, conidia ; *b*, oospores. (After W. C. Smith.)

This downy mildew, which flourishes in forcing-frames and glasshouses, was met for the first time in Europe in 1843–46, in North America 1876–86. It was not, however, until 1919–20, when several districts in California suffered heavy losses, that attention was paid to it.

Resistance to the disease varies considerably with the different varieties of lettuce (Jagger, 1924).

The fungus also attacks other plants belonging to the family *Compositæ*, in all about 126 different genera. Artificial inoculation experiments in Switzerland have shown that there is a sharp biologic specialisation in this species, as many as eleven different biologic forms having been distinguished: (1) f. sp. *Crepidis* on 10 species of *Crepis;* (2) f. sp. *Centaureæ* on 12 species of *Centaurea;* (3) f. sp. *Sonchi* on 4 species of *Sonchus;* (4) f. sp. *Picridis* on *Picris hieracioides;* (5) f. sp. *Lactucæ* on 5 species of *Lactuca;* (6) f. sp. *Cirsii* on 15 species of *Cirsium;* (7) f. sp. *Senecionis* on 11 species of *Senecio;* (8) f. sp. *Hieracii* on 19 *Hieracium* species; (9) f. sp. *Taraxaci* on *Taraxacum officinale;* (10) f. sp. *Lampsanæ* on *Lampsana communis;* and (11) f. sp. *Leontodontis* on *Leontodon hispidus.* Amongst the different specialised, or biologic, forms a certain number of minor morphological differences (such, for example, as the size of the conidia) could be detected. Inoculation tests made on plants of genera other than the one from which the conidia were obtained, yielded negative results throughout.

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Sclerospora graminicola (Sacc.) Schröt. var. *Setariæ italicæ* Trav. attacks the leaves of Italian millet (*Setaria italica*). The leaves become thickened and incurved; on the upper surface there is a scanty growth formed of thick-branched conidiophores with short side branches from which conidia are abstricted. Resting spores are produced in considerable numbers within the thickened parts of the leaves, and are finally liberated as a brown powdery mass. Often the inflorescence fails to grow out of the sheath (Traverso, 1902; Peglion, 1910; Weston, 1924). **S. macrospora** Sacc. attacks the male inflorescence, causing the formation of small, green leaves instead of the normal flowers. Conidia are only produced during the night, and they germinate as sporangia. Oospores are found in the leaf parts of the upper spikelets. The disease has been observed in Italy, and the same species of fungus is thought to cause a malformation of ears of wheat in Italy and France. The ears in such cases become bluish-green and thickened, sometimes they turn yellow ; they are partly surrounded by the uppermost leaves (Traverso, 1903 ; Arnaud. 1916 ; Weston, 1924).

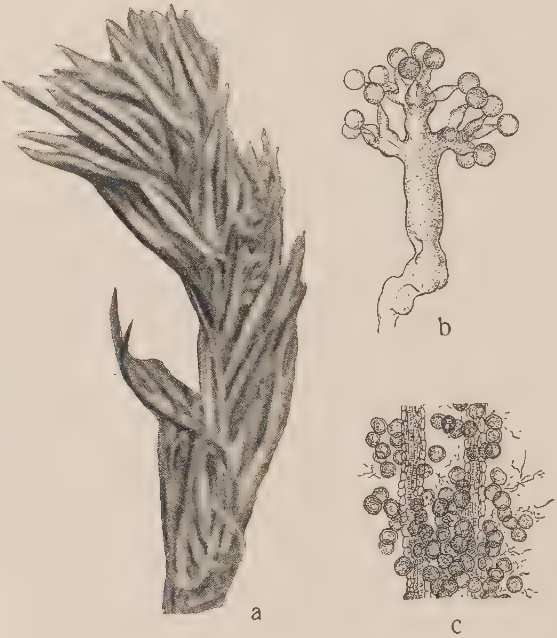

Fig. 86.
a, *Sclerospora macrospora* on male inflorescence of maize ; *b* and *c*, *S. graminicola* var. *Setariæ-italicæ* on Italian millet ; *b*, hypha with conidia ; *c*, piece of leaf with oospores. (*a*, after G. D'Ippolito and G. B. Traverso ; *b* and *c*, after A. Fischer.)

White Rust of Crucifers

Cystopus candidus (Pers.) Lév.

The fungi belonging to the genus *Cystopus* (or *Albugo*) are distinguished from those just described by the conidiophores and conidia not appearing on the surface of the leaf but being hidden beneath the epidermis as white, blister-like patches or sori. The conidiophores are short, unbranched, and clustered together, and from their tips chains of conidia are produced which are freed as a white powder when the epidermal covering ruptures. The fungus attacks all the organs—stem, leaves, leaf-stalk, cotyledons—of the host plant, making them appear as though sprayed with lime. The conidia usually produce several zoospores which have two cilia and germinate after a period of motility. Thick-walled, yellow to brown oospores, which are designed for over-wintering, develop in the infected tissues.

White rust attacks many wild crucifers, *e.g.* species of the genera *Capsella*, *Thlaspi*, *Sinapis*, etc., and it often appears on cultivated plants such as cabbage, swedes, turnips, rape, mustard, etc. The various forms of fungus found on the different host plants appear to be biologically specialised. As a rule this disease does not cause serious losses amongst agricultural crops.

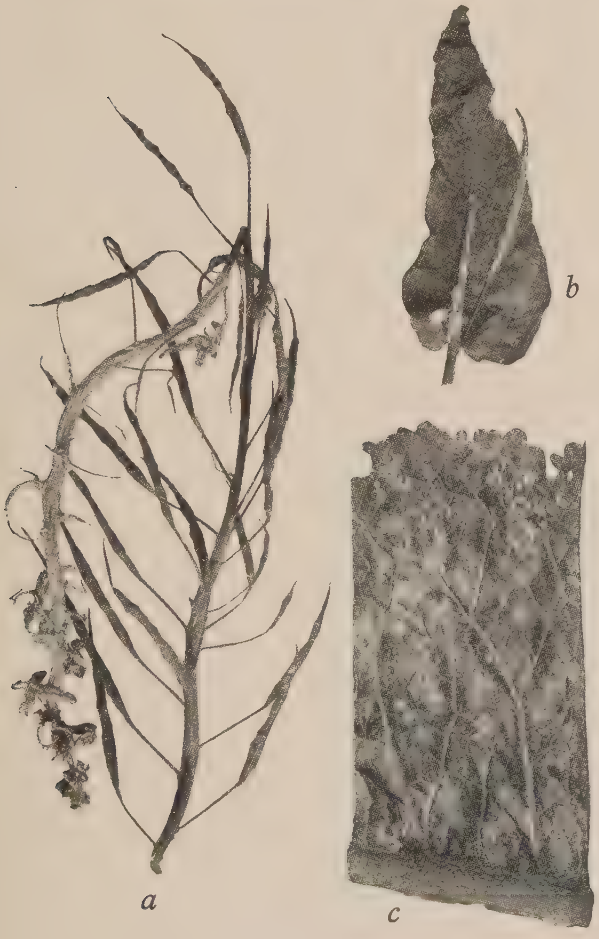

Fig. 87.—White Rust on Turnip.
a, inflorescence attacked by fungus ; *b*, diseased leaf in early stage ; *c*, in older stage. (Orig.)

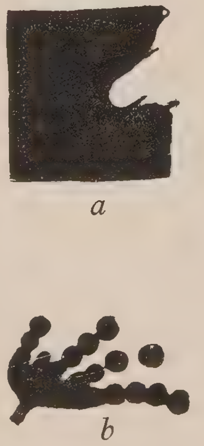

Fig. 88.
a, piece of leaf with two pustules of *Cystopus candidus*, one bursting ; *b*, conidiophores with chains of conidia. (After O. Kirchner and H. Boltshauser.)

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Cystopus Portulacæ Lev. forms irregular spots, with a whitish-yellow growth, on the upper side of the leaves of *Portulaca sativa* and *P. oleracea*. Oospores are found in certain leaves (Marchal, 1900; Montemartini, 1904). **C. Tragopogonis** (Pers.) Schröt on *Tragopogon porrifolius* and *Scorzonera hispanica*.

CHAPTER V

BASIDIOMYCETES

Reproduction in this class is by conidiophores or by basidia derived therefrom.

Ustilaginaceæ

These fungi are true parasites; the mycelium spreads through the tissues of the plant without, in the first place, noticeably interfering with normal growth. The fungus generally penetrates the flowering parts of the plant, forms spores there, and causes considerable damage. The spores appear as brownish-black, dusty masses in streaks on the sheaths and blades of the leaves, or they may completely fill the reproductive parts of the flower, the stamens and ovaries.

The spores are spherical, with a thick wall which is often verrucose or echinulate. As a rule the spores are separate, but sometimes several appear to have grown together; they either germinate at once or behave like resting spores and retain their viability for long periods, even as long as ten years it is said.

When a spore germinates it either produces a germ-tube which at once enters the host plant (if germination takes place on its surface) and there forms a mycelium, or else a short thick tube (basidium) is produced from which spores (basidiospores) are abstricted. If germination takes place in a special nutrient solution, *e.g.* a decoction of horse-dung, then budding, similar to that seen in yeast, takes place. The formation of bud cells continues as long as nourishment is available and can be continued almost indefinitely. By means of such cultures the very young parts of a plant susceptible to this form of fungus can be infected.

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Bunt or Stinking Smut—Tilletia

The spores are single, the surface rough or warty, rarely smooth. On germination, a short basidium develops which bears an apical whorl of thread-like basidiospores. These basidiospores are often joined in the middle to form pairs, and they either put out germ-tubes or develop bud cells which then germinate.

Bunt or Stinking Smut of Wheat

Tilletia Tritici (Bjerk.) Wint. and *T. lævis* Kühn

The wheat plants attacked by this disease do not differ markedly from healthy ones during growth. It is only after the seed has formed, and especially towards the time of ripening, that the glumes are seen to gape and some of the grains are found to be shorter and thicker than the normal. As a rule, all the ears on a plant and all the grains in an ear are affected, but sometimes diseased and healthy grains occur in the same ear. At the time of ripening, the bunted grains are grey in colour and filled with a brownish-black powder. They have a disagreeable odour resembling rotten fish, hence the name stinking smut.

The disease is caused either by *Tilletia Tritici*, which has spores with thick, ridged walls, or by *T. lævis*, which has smooth-walled spores; the former species is the commoner one.

The pericarp of a bunted grain does not burst of itself, but the sound and infected grains are harvested together, and it is only during threshing that numbers of the latter are broken up in the machine and the black spore-dust liberated. These spores adhere to the tuft of hairs, or "brush," at the end of the grain. Bunted wheat, as well as the flour made from it, has a fetid, fish-like smell. It is by means of these infected grains that the disease is carried to the crop in the following year, for when they germinate the bunt spores germinate also. The latter give rise on germination to a terminal cluster of long, narrow spores which either directly or by abstriction of conidia, produce germ-tubes by means of which the wheat seedling is infected. This method of transmission of the disease is known as "grain (seedling) infection."

There is also another way in which the young seedling can be infected, namely, by spores present in the soil. This "soil infection" must, in the light of experiments carried out in America from 1913 to 1921, be regarded as proved. It was found in these experiments, in which bunted ears of wheat were broken up, the spore-dust mixed with the soil in autumn, and the field sown with bunt-free seed, that the resulting crop contained more than 50 per cent. of bunted ears at harvest time. If, however, the bunt spores were destroyed by burning a thick layer of straw on the surface of the soil the crop remained free from infection. Coating the seed with copper sulphate solution led to a partial reduction in the degree of infection (Woolman and Humphrey, 1924).

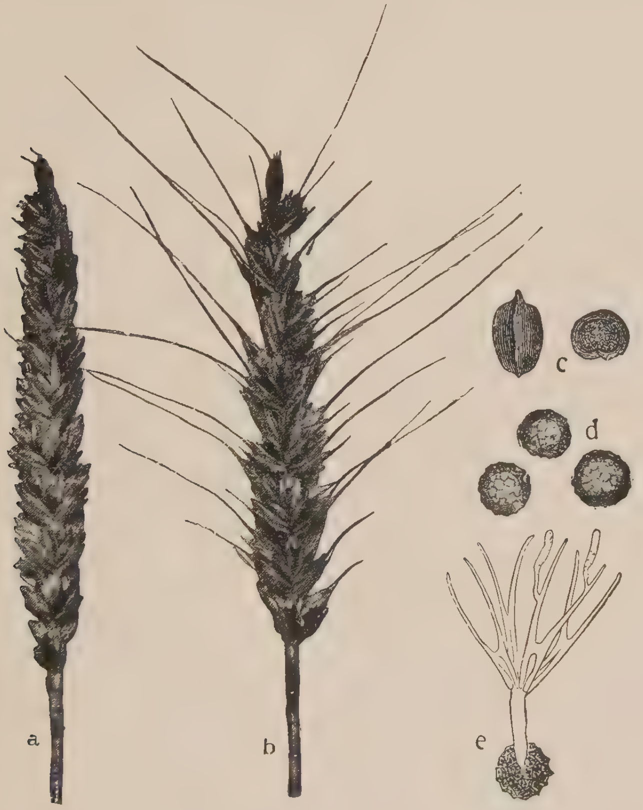

FIG. 89.—Bunt of Wheat.

a, bunted ear of wheat; *b*, bunted ear of awned wheat; *c*, two bunt-balls, one whole the other divided; *d*, three spores; *e*, germinated spore. (*a* and *b*, Orig.)

The viability and infective power of bunt spores depend largely upon the manner in which the spores have been preserved. In the open, under the most favourable conditions of temperature and moisture, they all germinate within 30–60 days and, consequently, after a period of this length no infection of the wheat seedlings is to be feared. Isolated spores which have over-wintered out of doors have, as a rule, been found incapable of germination in the following spring. Where spores fall on to dry ground in the autumn and the ground is then frozen during the winter, they remain viable and able to cause infection in the new year. Spores stored in a dry room (in a herbarium) have remained viable for as long as twelve years.

In America, it has been found that between the two fungi which cause bunt there are some other differences besides that of the structure of the spore wall. Thus, the haulms of wheat attacked by *Tilletia Tritici* are shorter, the ears broader, the spikelets grow closely together and are more expanded, the grains are plumper. Where the disease is due to *T. lævis*, the haulms are longer and develop narrower ears, spikelets and grains (Potter and Coons, 1918).

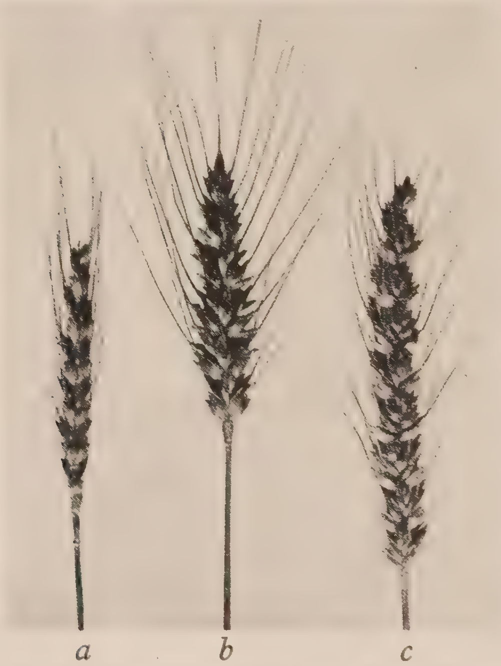

FIG. 90.—Ears of Wheat (Red Rock Winter Wheat). *a*, attacked by *Tilletia lævis*; *c*, by *T. Tritici*; *b*, healthy. (After A. A. Potter and G. W. Coons.)

Varieties of wheat, especially spring wheat, differ in their susceptibility to the disease, but the intensity of attack varies in different years. The temperature conditions at, or immediately after, sowing are of special importance. If at this period the temperature is low, the process of germination is delayed and consequently the time during which the seedling can be attacked is increased. Late sowing of winter wheat and early sowing of spring wheat favours, therefore, the progress of the disease (Hecke, 1900).

It has sometimes been thought that the feeding of bunted wheat to animals (horses, cattle, pigs, hens, and pigeons) leads to ill-effects, but no disturbance of the general health was observed when feeding tests were made, nor was any diseased condition of the internal organs to be detected (Baudys, 1921).

This disease can be traced back for more than 2000 years, to the time of the Greek savant Theophrastus Eresius, who lived between 371 and 286 B.C. It was long held to be a punishment of the gods or a consequence of unfavourable environmental conditions. The parasitic nature of the disease was first shown by M. Tillet, 1755, and the fungous origin demonstrated in 1807 by L. B. Prevost. In 1858, J. Kühn showed that the disease arose through infected seedlings. The use of copper sulphate for "dressing" the seed is due to H. Schulthess (1761), whilst about 100 years later, 1866, J. Kühn gave a recipe for this method of seed treatment. Recently a number of new materials and methods for the disinfection of the seed have been tested with more or less satisfactory results, and an account of some of these is given below.

Control Measures :

Seed should be obtained, if possible, from a crop which showed no signs of the disease. If such seed is not obtainable, then the following precautionary measures should be taken :—

1. *Washing the Seed.*—If whole bunt-balls are present in the seed corn they may be removed by pouring the grain little by little into a tub of water and stirring vigorously from time to time. The bunt-balls, which are lighter than the grain, float on the water and may be poured or skimmed off.

2. *Destruction of the Fungus Spores adhering to the Seed.*—As, in spite of washing, bunt spores remain attached to the grains, especially on the "brush" at the end of the grain, steps must be taken to get rid of them. To achieve this purpose several methods are available and a choice can be made between—

(*a*) *Disinfection with Copper Sulphate Solution.*—The sulphate of copper (bluestone, blue vitriol) is dissolved in water at the rate of ½ lb. to 10 gallons. The bluestone is placed in a piece of sacking and suspended in the water, where it gradually dissolves completely. The solution is then stirred and the seed wheat emptied into it ; the level of the liquid must be several inches above the grain and the whole well stirred several times. After 12–14 hours the liquid is poured off and the seed placed in a heap on a floor (that has previously been well washed with some of the copper sulphate solution) and sprinkled with milk of lime prepared by stirring 1 lb. of lime with 10 gallons of water. The seed is then spread in a thin layer to dry and when sufficiently dry it is sown. The sacks into which the wheat is placed after drying should first be disinfected by lying in a 2 per cent. solution of copper sulphate for 12 hours at least and then dried.*

(*b*) *Disinfection with Bordeaux Mixture.*—Copper sulphate (2 lbs.) is dissolved in water (5 galls.) in a wooden vessel. In another vessel, milk of lime is prepared by moistening 2 lbs. freshly burnt lime with a little water, and when the lime is slaked and has fallen to a fine powder, 5 gallons of water are added. The two liquids are then mixed by pouring them simultaneously into a third vessel. The Bordeaux mixture prepared in this way should be of a bright blue colour, it should not turn blue litmus paper red, and on standing a blue gelatinous precipitate settles out. A basket lined with coarse sacking is sunk in the Bordeaux mixture and into the basket the seed corn is poured. The seed is stirred up a few times and the bunt-balls floating on the surface are skimmed off. After 10–15 minutes, the basket is lifted from the liquid and the seed spread out to dry. By this treatment the grains are coated with a thin deposit which remains on them after sowing and exercises its fungicidal action after they have germinated.

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* The treatment with copper sulphate has the disadvantage that it somewhat lowers the percentage germination of the seed, especially when the threshing has been done with a machine. Seed corn treated with copper sulphate if not used for sowing cannot be used as food, as it is poisonous.

(*c*) *Disinfection with Formalin Solution.*—The solution is prepared by mixing 1 lb. of commercial formaldehyde solution (formalin) with 40 gallons of water. The seed is then poured in a thin stream into the diluted formalin and any bunt-balls or light grains that come to the surface are skimmed off and burnt. The seed is stirred vigorously several times and at the end of half an hour the liquid is run off and the grain spread out to dry in a very thin layer. Drying must be as rapid as possible so that the formaldehyde quickly evaporates and does not injure the germination of the seed. Seed corn disinfected in this way should not be kept for any length of time before being sown, as otherwise a reduction in germination results.

(*d*) *Disinfection with Uspulun.*—The use of Uspulun as a dressing for seed corn has been known for about twelve years. The active principle is mercury dichlorphenol. When the " steeping method " is adopted a ¼–½ per cent. solution of the Uspulun is prepared and the seed immersed in it for an hour and stirred several times. After steeping for the prescribed time the seed is spread out on a clean floor to dry. If the " sprinkling method " is used, the seed corn is watered with a ½ per cent. solution of Uspulun and turned over several times, so that each grain is thoroughly wetted. The treated seed is then covered with clean sacks and allowed to remain for 8–12 hours ; thereafter it is spread out to dry. The sprinkling method appears to be somewhat uncertain against bunt as failures easily occur. The steeping method is more reliable, especially if the ½ per cent. Uspulun solution is used. Seed corn that has been treated with Uspulun can, after being well washed, be fed in moderate amounts to poultry.

(*e*) *Disinfection with Germisan.*—Germisan, in which the active material is cyano-mercury-cresolate of sodium, is very reliable against bunt if the seed is free from bunt-balls. In the sprinkling method the amount required to treat 2 cwt. of seed is about 2–2½ galls. of ½ per cent. Germisan solution. Only half the amount of the solution is watered over the seed at first, and the rest is gradually added whilst the seed is being turned over with a shovel. This turning over should be continued for ½–1 hour according to the quantity of seed. The dressed seed is then covered with sacks, which have been previously soaked in the Germisan solution, and left for 6–8 hours, thereafter spread out in a thin layer to dry. For the steeping method, the Germisan solution is prepared at a strength of ¼ per cent. The disinfection by means of Germisan can be carried out several months before the seed has to be sown. Any seed remaining over after sowing can be fed along with other feeding-stuffs to poultry, up to 2 ozs. daily, or to pigs, up to 2 lbs. daily.

(*f*) *Dry Disinfection-dusts.*—Latterly the practice has developed of using Germisan and Uspulun in the dry form. A tight wooden barrel is half filled with the seed corn and the requisite amount of the dry powder—about 1 oz. to 20 lbs. of seed—is added and the barrel closed. The cask is then rolled about on the floor for 5 minutes to ensure thorough mixing. The barrel may, if preferred, be mounted on an axle and rotated by means of a handle. Dry disinfection of seed is very convenient and saves time and labour. The results, as far as present experience goes, compare well with those obtained by the wet treatment.

The numerous parallel experiments carried out in various places in Europe and America indicate that, up to the present, Germisan and then Uspulun can be recommended as seed disinfectants against bunt of wheat.

Amongst the other forms of smut fungi which attack cereals and grasses and fill the ovaries with a greyish-brown to black spore powder the following may be mentioned here: **Tilletia Secalis** (Corda) Kühn observed on rye in Russia, Germany, Austria, and Italy. **T. Pancicii** Bub. & Ran. on four-rowed barley in Serbia. **T. Hordei** Körn. on *Hordeum murinum* and *H. fragile*. **T. Lolii** Awd. on rye grass (*Lolium*) in Germany and Denmark. **T. decipiens** (Pers.) Körn. on bent grass (*Agrostis*) in Germany, Denmark, and Sweden. **T. Holci** West. on soft grass (*Holcus*) in Belgium and Denmark.

Other forms which belong here develop their spore-masses in elongated, erumpent streaks on the stems and leaves of grasses. Such a species is: **Tilletia striæformis** (West.) Oud., which attacks a large number of different grasses, *e.g.* species of the genera *Poa*, *Holcus*, *Festuca*, *Milium*, *Dactylis*, and *Phleum*. This fungus is found in most European countries.

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Smut Fungi—Ustilago

The spores are separate, verrucose, and smooth. On germination a short basidium is formed which divides into segments, each of which develops a basidiospore; the spores only rarely germinate directly.

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Loose Smut of Wheat

Ustilago Tritici (Pers.) Jens.

Loose smut differs from bunt in being recognisable as soon as the wheat begins to come into ear. The membrane surrounding the smut-masses bursts and the spores are at once disseminated by the wind. Soon nothing remains but the central axis to which some smut spores adhere, giving to it a black appearance. It was long thought that smut in wheat, barley, and oats was caused by the same fungus, but more recent investigations have shown that the smuts of the different cereals are separate species, each of which is only able to infect its own species of cereal plant.

The spores of the wheat smut are spherical, unicellular, and minutely verrucose ; they can germinate at once but remain viable only for a few weeks, so are not able to infect healthy cereal grains as do the spores of bunt.

The spores of this fungus are carried on the wind and reach healthy ears at the flowering time ; they adhere to the stigmas, germinate there like pollen grains, and send down their germ-tubes through the styles and infect the ovaries. This is termed "flower, or blossom infection," and the grain, as it develops, shows no outward sign of disease, save perhaps that is is smaller than the average.*

When such grains are used as seed the plants which they produce are smutted. The ears of a plant grown from infected seed are early filled with a black, dusty mass, which is spread by the wind and gives rise to fresh flower infections. The weather at the time of flowering influences the spread of the disease ; if heavy showers occur

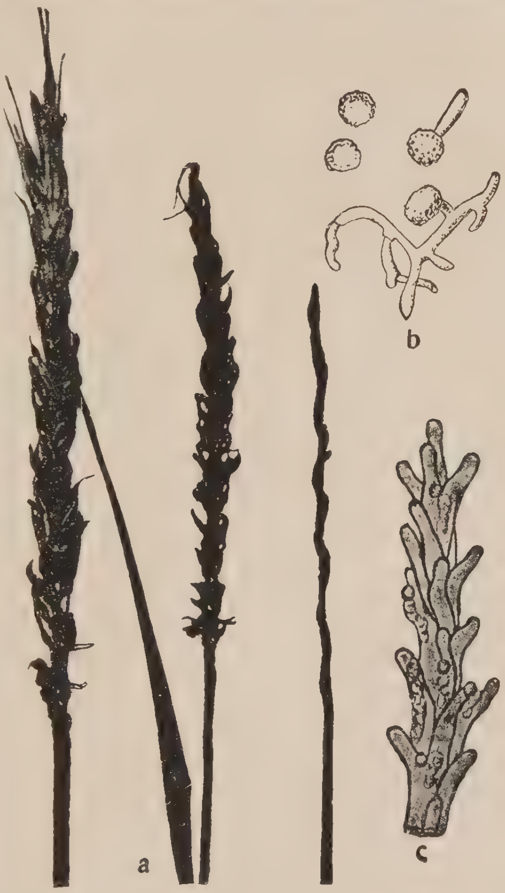

FIG. 91.—Loose Smut of Wheat.

a, three smutted ears, spores and glumes removed by wind from one on right, only axis remaining; *b*, ungerminated and germinated spores ; *c*, stigma of flower infected by spores. (*a*, Orig. ; *c*, after F. Falck.)

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* This mode of infection, although discovered as early as 1895 in Tasmania (Maddox) and 1896 in Japan (Hori), was made known in Europe through the investigations of Brefeld (1903) and Hecke (1904).

when the smutted ears are ready to free their spores, probably many are washed to the ground, germinate there, and never reach the stigmas of the flowers. Dry, calm weather, at the time when the spores are freed and the flowers are open, favours flower infection.

The fungus remains dormant in the grain as long as the latter is kept dry and there forms a resting, or intraseminal, mycelium. If, however, the grain is soaked for a few hours in tepid water the mycelium becomes active and is then sensitive to higher temperatures, a fact made use of in the hot-water treatment used in disinfecting seed grain.

In addition to flower infection there is probably also a grain infection, as in the closely related *Ustilago nuda* (see below), seeing that such disinfectants as formalin, Uspulun, etc., have proved of value in controlling the disease.

Control Measures :

(1) Seed should not be used from crops in which smut has been found at flowering time, as probably many flowers have been infected and the grains from them will produce smutted plants. (2) If the seed comes from an infected crop it should be treated as follows : A sack is filled two-thirds full of seed and soaked for 4–6 hours in water at a temperature of 20°–30° C., the sack is then put into water of 50°–54° for 20 minutes. If this hot-water treatment cannot easily be carried out, the seed may be disinfected with formalin solution or some proprietary " steep " such as Uspulun or Germisan.

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Loose Smut of Barley

Ustilago nuda (Jens.) Kell. & Sw.

This disease of barley is very similar to *Ustilago Tritici*. It appears at the beginning of the flowering period. The spores are at once disseminated by the wind and many reach the healthy barley ears when they are in flower. Some of the spores adhere to the stigmas of the flowers and there germinate, sending down the germ-tubes and infecting the ovaries. The infected flower develops its seed normally, but within the latter is carried the dormant or intraseminal mycelium which, in the following year, infects the plant and causes the formation of smutted ears.

The spores are spherical with verrucose walls. When a spore germinates, a long-jointed and branched germ-tube develops and infects the pistil.

Latterly it has been shown in the United States that the spores which have been deposited between the glumes at the flowering period give rise to a grain infection, which explains why steeping in formaldehyde, Uspulun, Germisan, etc., has been effective in disinfecting seed grain. The fact that certain sorts of barley suffer more than others from the fungus is probably due to differences in the structure of the flowers; in one sort the pistil may be more open to infection than it is in another. Dry weather at the flowering period favours flower infection, whereas heavy showers of rain wash a lot of spores to the ground where they are not harmful.

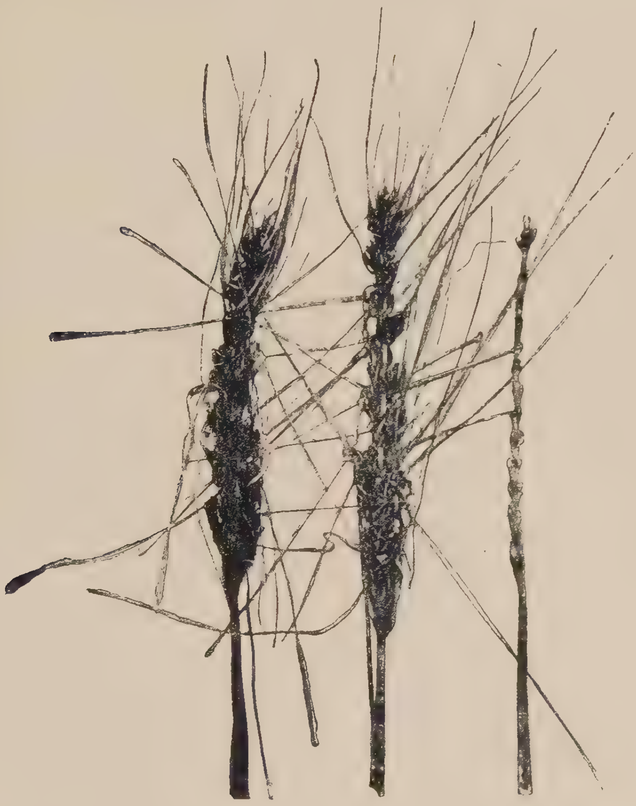

Fig. 92.—Loose Smut of Barley.

Three smutted ears, from one of which brand spores and glumes have blown away, leaving only the axis. (Orig.)

For the control of the disease the same measures should be adopted as for smut of wheat.

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Covered Smut of Barley

Ustilago Hordei (Pers.) Kell & Sw.

Covered smut of barley differs in several important respects from loose smut. At flowering time, ears of barley attacked by the disease have very little to distinguish them from healthy ones. A few weeks later, however, the infected ears are of a dark colour and the spikelets are changed into broad, swollen structures in which three points or teeth can be distinguished, the middle tooth corresponding to the middle flower, and the side teeth to the lateral flowers. The silver-grey membrane surrounding the smut spores remains intact up to the time when the barley is ripe, or it may show a few small cracks. The spore-dust is thus not disseminated during the flowering period of the barley, and so flower infection is impossible. The closed, awnless, smutted ears are harvested along with the infected ones, and it is not until the crop is threshed that the spore-dust is freed and carried to the clean grain. When such infected grain is used for seed, the spores carry the disease to the seedling, which, when it grows into a plant, produces smutted ears. This grain, or seedling, infection is similar in type to that which occurs in bunt of wheat.

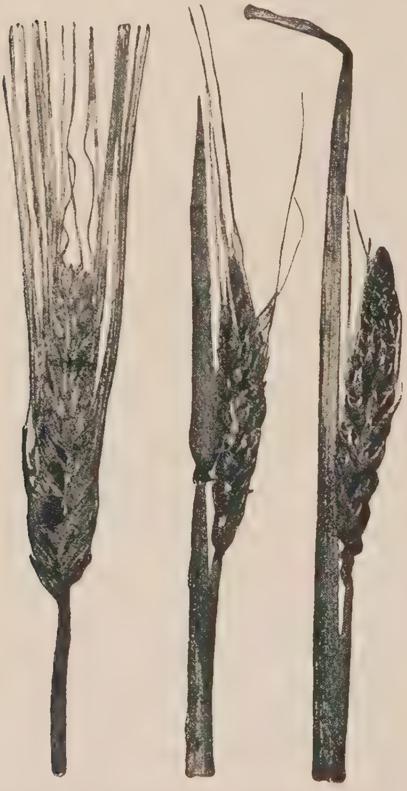

Fig. 93.—Covered Smut of Barley. (Orig.)

The spores of the fungus are smooth and somewhat larger and more angular than those of the loose smuts. When they germinate they produce a short germ-tube which gives rise to spores, and these increase by budding.

Control Measures :

These are the same as those taken in the case of smut of wheat. It is not permissible, however, to use copper sulphate as a disinfectant, as it reduces the germination of the seed.

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Loose Smut of Oats

Ustilago Avenæ (Pers.) Jens.

This smut is the commoner of the two smuts which affect oats and is similar to the loose smuts of wheat and barley. The infected panicles can be distinguished at the time that the oats are shooting into ear, as they are more compact than the healthy ones. Sometimes all the spikelets of the panicle are infected, or again healthy and diseased spikelets may be present, the healthy ones usually being found at the top of the panicle.

The spikelets that have been completely destroyed by the disease are rounder in shape and are filled with a black, powdery mass. Partially diseased spikelets retain their natural shape more or less and only the lower part is filled with the smut spores.

The dissemination of the spores continues for a longer time than is the case with wheat and barley, and takes place from the beginning of the flowering period up to the time when the grain is ripe; when the crop comes to be harvested most of the spore-dust has been dispersed. In spite of the early dispersion, flower infection rarely, if ever, occurs, but spores settle between the glumes of the open flower and are retained there, or the spores adhere to the grain when it has been formed.

The disease is caused by *Ustilago Avenæ*, which has spherical, verrucose spores. These spores usually germinate by a short germ-tube, from the tip and joints of which bud cells are formed which are able to infect young seedlings. Germination takes place in warm weather and the spores remain viable for several years. With this form of smut the infection is always of the grain, or seedling, type.

Often 30 per cent. of the crop—in extreme cases 60 per cent.—is destroyed, and it is rare to find an oat crop entirely free of the disease. Even a slight attack is dangerous as it may lead to a severe one in a future year. Dry, calm weather at the time when the oats are in flower favours the entrance of the spores, and warm, damp weather at seeding time leads to the infection of the germinating seed. Both of the conditions mentioned give rise to a serious outbreak of disease.

Control :

The same measures apply as for bunt of wheat and covered smut of barley. It is not necessary, however, to wash the seed grain as the liquid would not reach the spores which lie between the grain and the glumes, having got there at the time of flowering. Copper sulphate is not suitable as a disinfectant, as it reduces the germinating power of the oats.

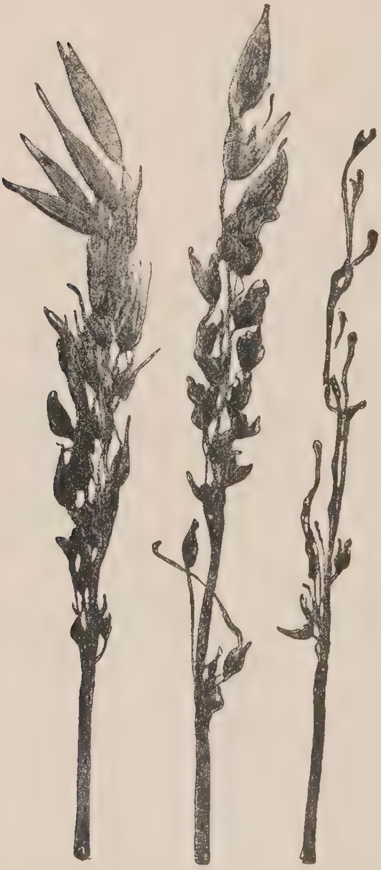

FIG. 94.—Loose Smut of Oats. Three smutted panicles; from one the spores and glumes have blown away. (Orig.)

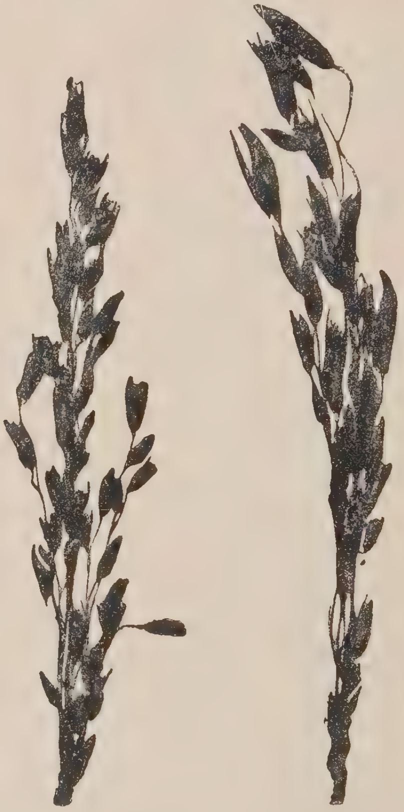

FIG. 95.—Covered Smut of Oats. (After O. Appel.)

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Covered Smut of Oats

Ustilago Kolleri Wille.

At first, neither the panicles as a whole nor the individual spikelets show any outward sign of infection. About the time of ripening, however, a black mass can be seen shining through the glumes which, as a rule, remain closed until the grain is ripe. The spores are not liberated until the grain is threshed; they are smooth and dark brown in colour, and adhere to the oat grains if left undisturbed. It is by these spores that the young seedlings are infected (Wille, 1893).

Smut of Maize

Ustilago Maydis (DC.) Tul.

This disease attacks the male and female flowers, stem, leaves, and even roots of maize. The grains—especially those at the tip of the ear or cob—are changed into smut receptacles or sori, which at first are covered by a greyish-white membrane. These sori are much larger than the grains themselves and together may produce tumour-like growths or galls of considerable size. The contents of the sorus is at first brownish-black and sticky, but when the sorus bursts a dry black powder is liberated and dispersed by the wind. On the stems and leaves hard, shining, pale grey, irregularly-shaped lesions are produced which may be in the form of galls (as large as a small child's head in some cases) or small wart-like blisters; in both cases the interior is filled with black spore-dust.

When placed in water, the spores do not always germinate at once, after they have become free, but may remain dormant until the following spring. In nutrient solutions and in decoctions of dung or freshly-dunged soil, the spores germinate at once very actively and increase by budding. When the spores germinate they give rise to a branched germ-tube from which spores are produced, and these are carried on the wind. If the spores reach the neck of the root of a young seedling or the growing point of an older plant where young leaves or flowers are being formed, they adhere, infect the plant, and in about three weeks give rise to one or other kind of malformation. The fungus does not spread from the point of infection throughout the whole plant—as is the case with the smut fungi already described—but remains near to the original point of infection. Each group of growths, whether in the shape of tumours or warts, is therefore the result of a single infection and has no connection with other groups on the same plant. As the spores are able to live for a long time—it is said for several years—and as on the maize plant young organs are present well on into the summer, it is easy to see that infection may take place throughout the greater part of the growing period. The appearance of the disease in the field is first noticed when the maize plants have grown to a height of 3 feet or so.

This smut is known in all maize-growing countries. It sometimes affects only a few plants (2–3 per cent.), but it can be more general and very destructive, as many as 30 per cent. being attacked.

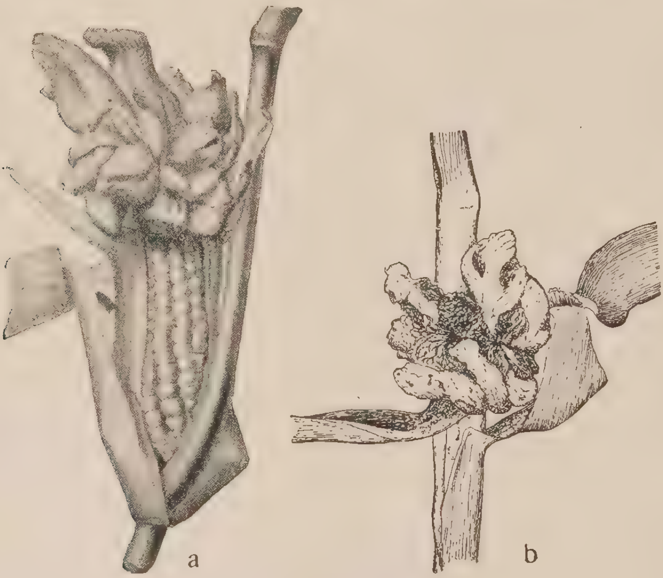

Fig. 96.—Smut of Maize.
a, cob with upper grains replaced by brand spores ; *b*, gall in axil of leaf.
(*a*, after O. Brefeld ; *b*, after A. S. Hitchcock and B. S. Norton.)

Control Measures :

(1) All diseased plants should be collected and destroyed as soon as noticed and before the spores are freed. (2) Fresh dung should not be applied to maize, as it has been shown to favour the growth of the fungus in the soil and also the infection of the plant. (3) Refuse from infected plants must be burnt and not put on the manure heap.

Two other species of smut fungi—**Ustilago Fischeri** Pass. and **U. Reiliana** Kühn—are found on maize. They are less destructive than the fungus mentioned above and are only found on the female inflorescences. The first of these fungi attacks the central axis of the cob and in this way causes many of the grains to be small and undeveloped. Both forms of smut have been observed in Italy, and recently the latter has been found in the United States (Dana, B. F., and Zundel, G. L., 1920), not only on maize but on millet, where it causes the whole panicle to be changed into a single smut sorus.

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Sorghum Smuts

Ustilago Sorghi (Link) Pass., *U. bulgarica* Bub., and *U. cruenta* Kühn.

The first-mentioned species, *Ustilago Sorghi*, only attacks the flowers, the ovaries of which grow out to long, smooth, sack- or horn-shaped structures. These bodies extend beyond the glumes and are covered by a thick, brown membrane. They contain a brown spore-dust which surrounds a central column. The axial parts of the panicles develop normally and there is no shrivelling or malformation of the panicles. The smut attacks *Sorghum vulgare* and *S. saccharatum* and is known in all countries where sorghum is grown.

Ustilago bulgarica is closely related to the above and has been observed on *Sorghum vulgare* in Bulgaria. It is distinguished from *Ustilago Sorghi* in that the horn-like smut sori are pitted or furrowed on the surface and covered with a grey membrane; the spores are light olive-brown in colour and not very dusty.

Ustilago cruenta occurs on both varieties of sorghum mentioned above and attacks all parts of the panicle. It is chiefly seen as small, brownish-red, rounded or elongated outgrowths which sometimes coalesce and give rise to large tumours. In severe attacks, the branches of the panicle are shortened, thickened, and twisted, causing the whole to be misshapen. At times lesions are also found on the stalk, immediately below the panicle. The ovaries are only rarely attacked, but when this occurs they grow out to long, horn-shaped, reddish-brown sori. This disease is found wherever sorghum is grown and in many places causes severe losses.

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Ustilago Panici-miliacei (Pers.) Wint. appears on *Panicum miliaceum*, *P. Crus-Galli*, and other species of this genus. The fungus only attacks the inflorescence, which is changed to a compact, firm, cob-like structure covered with a whitish membrane and enclosed in the sheath formed by the youngest leaves. The inside of this " cob " is filled with spore-dust together with vestiges of the rachis. The white membrane surrounding the spore-mass gradually bursts and the black dust is freed. The disease has been observed in Italy, France, Germany, and the United States (Hecke, 1902, 1903).

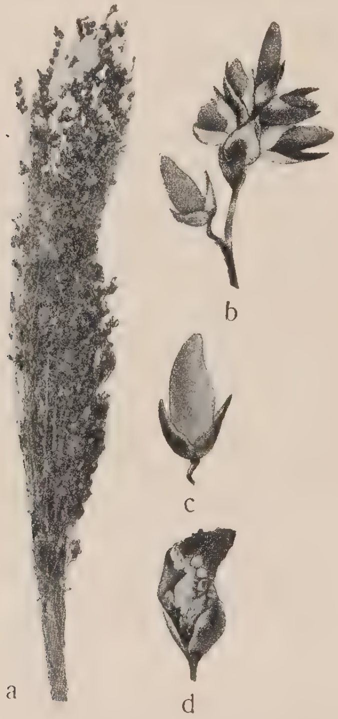

Fig. 97.—*a–c*, *Ustilago Sorghi*. *a*, whole panicle ; *b*, branch of panicle ; *c*, single flower with spore mass ; *d*, *Ustilago bulgarica*, single flower with spore-mass. (After F. Bubák.)

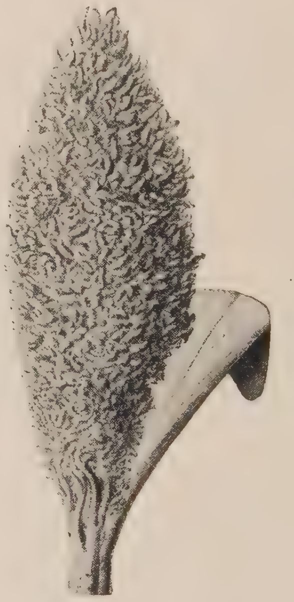

Fig. 98.—*Ustilago cruenta* on sorghum. (After O. Brefeld.)

A number of other species of smut fungi attack *Panicum miliaceum* and other species of the same genus in Southern Europe and adjoining countries. One of these fungi is *Ustilago Rabenhorstiana* Kühn, which destroys the panicles as well as the stem below. *Ustilago Digitariæ* (Kze.) Rabh., on the other hand, only attacks the flowers.

Smut fungi have also been observed on such species of millet as *Setaria italica, S. viridis, S. germanica.* The best known is **Ustilago Crameri** Körn., which destroys the seeds by filling them with a black spore-dust. This disease causes considerable damage in Southern Europe; in Austria and Hungary sometimes one-third or more of the millet grain is destroyed.

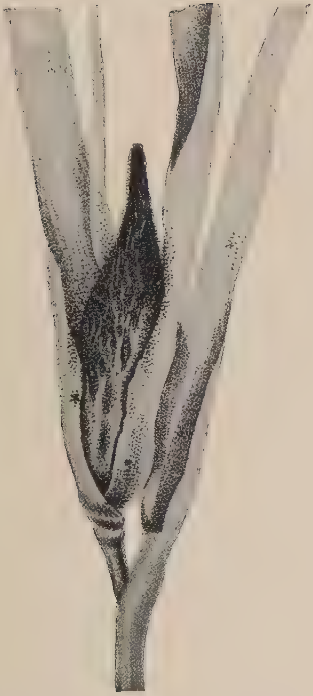

Fig. 99.—*Ustilago Panici-miliacei.* (After E. v. Jaczewski.)

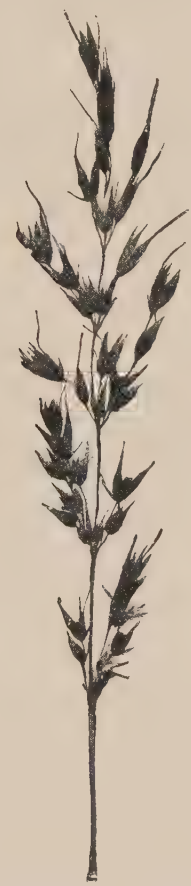

Fig. 100.—*Ustilago perennans.* (Orig.)

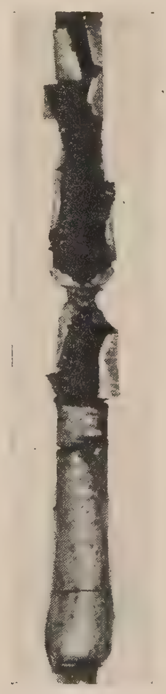

Fig. 101. — *Ustilago grandis.* (Orig.)

Amongst the smut fungi which attack grasses and destroy the seeds the following may be mentioned: **Ustilago perennans** Rostr. on *Avena* *elatior* and **U. bromivora** Fisch. & Waldh. on *Bromus avensis*, *B. mollis*, and others.

In addition, there are some forms of loose smut that develop their spore-dust in the leaf and stem of various grasses. Such a form is **Ustilago longissima** Low. which gives rise to long, parallel, open streaks filled with olive-green spore-dust on *Glyceria aquatica*. As a rule, these infected shoots do not produce ears. If smutted grass is fed in a green state to cattle it may cause illness or even death; the characteristic symptoms are diarrhœa, shivering, and paralysis. When, however, the grass is made into hay it does not appear to be dangerous (Eriksson, 1900).

Other forms of *Ustilago* on grasses are **Ustilago hypodytes** (Schlecht.) Fr., which forms a brownish-black coating on the stems and sheaths of *Glyceria fluitans*, *Elymüs arenarius*, *Phragmites communis*, and other grasses, and **Ustilago grandis** Fr., which produces finger-thick swellings filled with a black dust on the uppermost joints of the stalks of *Phragmites communis*.

Ustilago violacea (Pers.) Fuck. The spores are formed in the anthers of a large number (about 70) of the *Caryophyllaceæ* such as *Dianthus*, *Melandrium*, *Viscaria*, *Silene*, *Saponaria*, *Stellaria*, etc., which are attacked by this fungus. The infected anthers are swollen, the sori porous and of a violet colour. The spore-dust is usually transported in the night (8–11 o'clock), by insects having a long proboscis, to the stamens and styles of the flowers (flower infection), perhaps also at times by the wind to seedlings and young shoots. After infection, the anthers are filled with spore-dust (Werth, 1911). The disease does not appear to be spread by infected seed. Several specialised forms of this fungus are distinguished; for example, one on *Dianthus superbus*, another on *D. deltoides*, a third on *Saponaria officinalis*, a fourth on *Melandrium album* (this form has been artificially transmitted to *Agrostemma Githago* and *Silene noctiflora*), and others (Zillig, 1921).

Very closely related to the above is **Ustilago Betonicæ** Beck, which fills the anthers of *Betonica alopecurus* and *Salvia pratensis* with violet-brown dust (Kirchner, 1923).

Ustilago Vrieseana Vuill. gives rise to small, hard galls on the lower nodes of the stems of various species of the genera *Eucalyptus*, *Myrtus*, and *Callistemon*. From these galls, twigs in the form of witches' broom are put out; Brownish-violet spores are contained in the galls. The disease has been observed in Holland and France (Vuillemin, 1894, 1895).

Urocystis

The spores are compound, each spore-ball consisting of about ten spores one or more of which are larger and darker and lie in the centre, whilst the surrounding ones are smaller and lighter. On germination each of the central darker spores gives rise to a basidium which develops basidiospores terminally.

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Stripe Smut of Rye

Urocystis occulta (Wallr.) Rab.

The sori arise on the stems and leaves in the form of long streaks which, at first, are covered by the epidermal tissue and show a dark grey colour. When the covering bursts, the black spore-dust becomes visible. The infected stems are more or less misshapen and stunted. Usually the ears do not mature or are devoid of grain. As a rule all the stems of the plant are attacked.

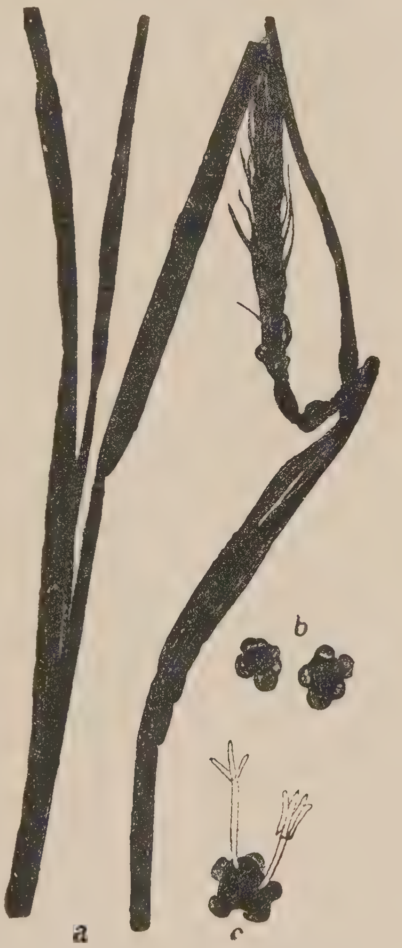

Fig. 102.—Stripe Smut of Rye.
a, smutted stems; *b*, spore-balls ungerminated; *c*, germinated spore-balls. (Orig.)

Control Measures: the same as against bunt of wheat.

Urocystis Tritici Körn. Flag Smut of Wheat. The disease is closely related to rye smut and has been known in Australia since 1868. It has spread there very considerably and is now the most destructive disease of wheat, each year reducing the crop by at least 3 per cent. and sometimes by as much as 70 per cent. The disease is also known in North

America, Japan, China, Italy, and Spain. The spores usually germinate badly but under certain stimulating conditions germination is somewhat better (Noble, 1923, 1924; Griffiths, 1924).

Urocystis Bolivari Bub. & Frag. is found in Spain on *Lolium perenne* (Bubák, 1922).

Onion Smut

Urocystis Cepulæ Frost

This fungus produces leaden-grey sori in the leaves, especially the cotyledon leaves, and in the young onion structures of *Allium Cepa*. The sori are formed under the epidermis, and finally become irregular, open pustules from which a blackish-brown, dusty spore-mass appears. The spore-balls have a central, spherical, reddish-brown main spore around which are grouped numerous (15–40), smaller subsidiary spores of a lighter colour. Only the central spore is capable of germination. Young onion seedlings attacked by the fungus do not develop but remain stunted, and finally dry up or decay.

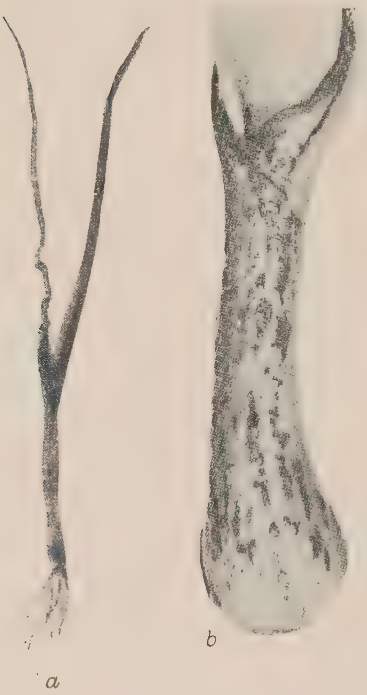

Fig. 103.—Onion Smut. *a*, young diseased plant; *b*, old diseased plant. (*a*, after H. C. Müller and E. Molz.)

The central main spore germinates at once and the germ-tube infects the onion seedling, particularly the cotyledons, provided the young plants have not reached a length of 7–8 cm. Seedlings that have grown beyond this stage are immune even when growing in infected soil. After an incubation period of 5–10 days, new pustules break out. If, however, the germ-tube as it grows finds no onion seedling at hand it develops into a mycelium that is capable of living saprophytically in the soil, probably for several years.

In the United States, where the disease is very common in certain districts and much feared, the influence of weather and soil conditions upon the disease has been studied and an explanation given why in the northern districts, from New York to Oregon, severe losses are experienced, whilst in the south—Texas and Louisiana—it does not occur, although onion-growing is common and there is the same risk of infection being carried. Apparently the difference is due to the fact that in the north onion seed is sown in spring in the open, whilst in the south it is sown in plots in late autumn and the young seedlings planted out in spring. Thus in the south, the danger of infection at a time when weather conditions are unfavourable is reduced and the seedlings if attacked are able to grow away from the disease.

Onion Smut has been known in America (Connecticut River Valley) since 1860; it caused much loss in the 'seventies and is to-day the widest-spread onion disease. In Europe it was first observed in France in 1879 (Cornu, 1897), and is said to have been known in Germany about this time, but the first serious outbreak was in 1914 (Müller and Molz, 1917). It has been known in England since 1915 and was found in 1919 in three different localities, in one case where American seed had been used (Cotton, 1921). In Switzerland the disease first appeared in 1924, on onion plants raised from French seed (Müller-Thurgau and Osterwalder, 1924).

Preventive and Control Measures :

(1) Diseased plants should be carefully collected and burnt before the spores are liberated. (2) Infected soil should not be sown with seed for several years, but the plants should be grown from sets or from seedlings, 7–8 cm. long, which have formed the first seed leaf and so are immune. (3) When onions are grown on a small scale, non-infected ground should be chosen. (4) If onions are grown on a large scale, formalin solution (0·4 per cent.) should be applied when the seed is being drilled (Walker, 1920; Jardine, 1922; P. J. Anderson and Osmun, 1923; Gregory, 1923).

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Urocystis Galanthi Pape produces hard, blister-like sori on the leaves, leaf sheaths, and flower sheaths of the snowdrop (*Galanthus nivalis*). These sori are of a leaden-grey colour, and in them the spore-masses are embedded. The spore-balls are composed of 1–3 dark brown main spores and numerous peripheral spores of a lighter colour. The mycelium seems to be perennial in the bulb. The disease was observed in 1921 near Berlin (Pape, 1923).

Closely related are **Urocystis Colchici** (Schlecht.) Rab. on *Colchicum autumnale, Muscari comosum*, etc., **U. Gladioli** W. G. Sm. on *Gladiolus nanus*, **U. Ornithogali** Körn. on *Ornithogaliun umbellatum*, and **U. Leucoji** Bub. on *Leucojum vernum*.

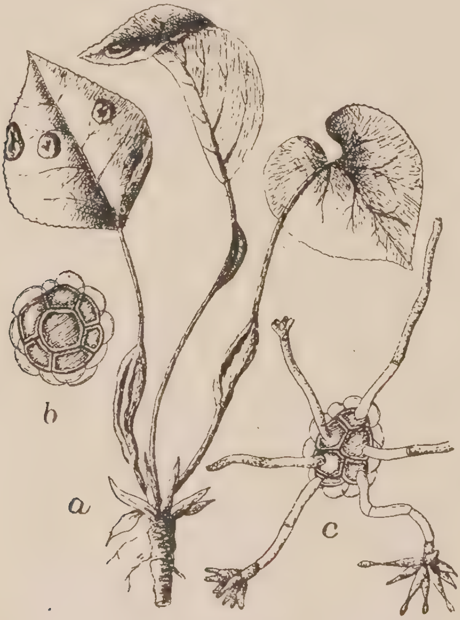

Fig. 104.—*Urocystis Violæ* with spore-balls. (After T. Ferraris.)

Fig. 105.—*Graphiola Phœnicis.* (Orig.)

Urocystis Anemones (Pers.) Wint. forms large, horny sori on the lower surface of the leaves and on the leaf-stalks of *Anemone nemorosa*. Closely related hereto is **U. Hepaticæ-trilobæ** DC., which produces similar elongated sori on the leaves and leaf-stalks of *Anemone hepatica*. Both the above forms occur in most northern and mid-European countries as well as in the United States.

Urocystis Violæ (Sow.) Fisch. v. Waldh. The sori are in the thickened swellings on the petioles, leaf blades, and stalks of *Viola odorata* and other species of *Viola*, such as *V. hirta*, *V. cornuta*, etc. Shrivelling and deformation of the infected leaf-stalk is the regular consequence of the disease. The sori are black and powdery and the spore-balls contain 4–8 main spores surrounded by a layer of lighter-coloured sterile cells. The mycelium appears to over-winter in the root-stock.

Closely related is **U. Kmetiana** P. Magn., which fills the ovaries of *Viola tricolor arvensis* with a black powder; it has been observed in Hungary and France (Poirault, 1915).

Entyloma Ranunculi (Bon.) Schr. attacks the petiole of *Helleborus niger* preferably at the base at soil level and at the tip where the leaflets emerge. The parts that are attacked shrivel up or rot. The infected petioles together with the leaflets soon die. The disease was observed in Savoy (Arnaud, 1919). **E. Œnotheræ** March. & Stern. forms large, round, red to brown spots, which often coalesce, on the leaves of *Œnothera Lamarckiana* and *Œ. biennis*. These spots are covered on both sides with tufts of conidiophores, which emerge from the stomata. The conidial stage of the fungus was formerly named *Ramularia Œnotheræ biennis* Ivan. Double-walled chlamydospores are found in the tissues of the leaf below the tufts, they have arisen from intercellular hyphæ and germinate after a resting period (Marchal and Sternon, 1924).

Graphiola Phœnicis Poit. produces on the leaves of the palm, numerous black, round, or elongated sori which contain a pale yellow spore-mass. The spores are very small, spherical in shape, and on ripening give rise to rounded conidia which germinate readily. This fungus is endemic in palm-growing countries (Algiers, Barbadoes, etc.) and has sometimes appeared on *Phœnix dactylifera* in European greenhouses, thus in Denmark (1900–02, 1922) and in North Sweden (Korsnäs, 1909). To control the disease it is recommended to remove the infected leaves and to place the plants in a cool, well-ventilated place.

CHAPTER VI

UREDINACEÆ—RUST FUNGI

THESE fungi are true parasites, and the vegetative system, the mycelium, spreads as a network of hyphæ between the chlorophyll-containing cells of the organs that are attacked. As a rule only short branches of the mycelium (haustoria) actually enter the cells, but in some exceptional cases a much-branched network is found there. The cells are usually not destroyed by the mycelium but retain their normal shape for a long time. They are, however, ultimately pressed together by the growing mycelium and their contents—cell walls, chlorophyll granules, starch grains, etc.—is killed by the fungus and used up. Finally, a coherent fructifying organ, the hymenium, is formed, from which long branches arise, and from these branches spores are abstricted. As a rule, the epidermal layer covering the hymenium ruptures and an open wound, filled with yellow or brown dust, arises. In certain cases the epidermal covering remains intact and is spread as a transparent membrane over the spore-group.

There are usually several types of spores present in the rust fungi; in summer the summer spores (uredospores), which are yellow or yellowish-red, one-celled, verrucose or spiny and able to germinate at once. The germ-tubes find entrance through the stomata and after a few weeks new pustules are formed. Several such spore-generations can follow in succession during the course of the summer.

Autumn, or winter, spores (teleutospores) are formed from the same mycelium and are brown or black, one- or many-celled, and with thick walls; they may germinate at once, but they usually function as resting spores and do not germinate until the following spring. When these spores germinate a short basidium divided into segments is put out, and from each segment a basidiospore (sporidium) is separated; these basidiospores are transported by wind. When such a spore reaches a living leaf, or other part of the plant, of a species which it is capable of infecting, it pushes its germ-tube through the wall of the epidermis, grows on inside the organ, and gradually gives rise to rust pustules on the surface of the infected organ. The germ-tube may enter the same species of host plant as that on which the spore was produced, or a closely related one, the fungus then being termed autœcious. If, however, the fungus invades a plant of a widely different species from that on which the spore originated it is termed heterœcious.

By the entry of the fungus into the barberry leaf the sporidium puts out a short, beak-like germ-tube which produces an extremely fine style-like structure which penetrates the wall of the epidermal cell (Waterhouse, 1921). Varieties of Barberry and Mahonia with thin external epidermal walls are most susceptible to rust. All varieties become immune with increasing age (Melander and Craigie, 1927).

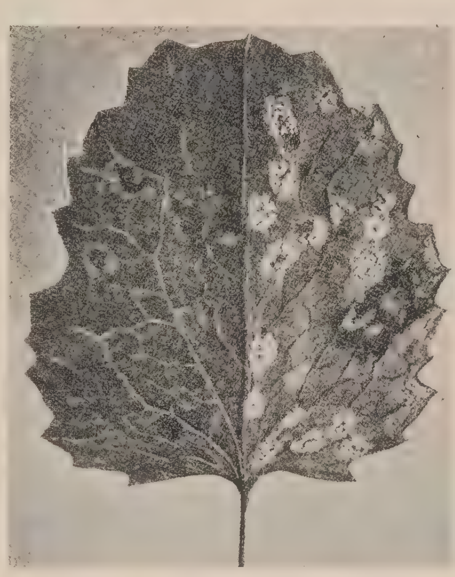

FIG. 106.—Under side of a Barberry Leaf. The right half with well-mixed pycnidial-nectar and abundant æcidia, the left half with separately stirred, not mixed, pycnidial-nectar, no æcidia. (After J. H. Craigie, 1927.)

The stage of the fungus which arises when there is a change of host plant, differs markedly from the uredo- and teleuto-stages described above and is termed the cluster cup or æcidial stage, and in it are usually two forms of fructifications. The first form occurs as pycnidia, which are very small, flask-shaped bodies arranged in groups and embedded in the leaf; they contain a large number of narrow, rod-shaped pycnoconidia (spermatia). The second form, which generally appears on the opposite side of the leaf, consists also of small groups of closely-aggregated cups with reflexed, ragged edges (æcidia) or else naked groups of spores (cæoma).

The function of the pycnidial stage in the development of rust fungi has not been satisfactorily explained, but recent investigations (1927) throw considerable light on the question. From experiments carried out in the Dominion Rust Research Laboratory in Winnipeg (Canada) it was discovered that two kinds of pycnidia occur. When the nectar, containing pycnoconidia, from any one pustule is mixed by means of a scalpel with the nectar of several other pustules, æcidia are rapidly formed in most of the pustules, whereas when the nectar is not mixed, most of the pustules do not develop æcidia. Out of doors, flies mix the nectar from the different pustules (Craigie, 1927).

The æcidiospores are arranged in chains and, as a rule, germinate at once. If they alight upon the corresponding uredo-bearing species of host plant they can produce pustules with summer spores within 8–10 days.

The teleutospores are the most important of the spore-forms, as by means of them the fungus can generally hibernate until the following year. The systematic arrangment of the rust fungi and the nomenclature of the genera are also based on these spores. The different species are, however, named after the host plant.

Some of the rusts (heterophagus species) can live on several species of closely-related host plants, but others (isophagus species) are confined to one species of plant.

With some fungi there is a " specialisation " of parasitism, that is to say, in the same species there occur several biologically different races—specialised forms (f. sp.). These are of similar outward appearance but are distinguished by internal biological characteristics, which are shown by the circumstance that each form lives on its own—either one or more—species of host plants.

Cases have been noted in which the reappearance of a disease in the following year could not be explained by reference to the winter spores. It has been suggested that in such cases an over-wintering mycelium is present in the stem or rhizome of the host plant, or there may be hibernating uredospores. The first of these suggestions has hardly been disputed, but the presence of a hibernating uredo-stage as a link in the life-history of the rust fungi has raised doubts. It is certainly true that now and then and with different rusts, hibernating uredo-pustules had been observed and that in them viable spores were found in the spring—March, April, or May. No proof has, however, been given that the normal, fresh outbreak of disease in summer has its origin in such accidental pustules. A critical survey of the literature on this subject leads to the conclusion that available evidence does not adequately support such a view.

This opinion has recently received support from some experiments made in Lincoln (Nebraska) during the years 1920–25 with the object of obtaining further information concerning the over-wintering of the uredospores of *Puccinia graminis Tritici*. The field observations showed that uredospores collected after the month of January were no longer viable as they could not be made to germinate. The question of the over-wintering of these spores can therefore, as regards Lincoln, be answered in the negative (Peltier, 1926 ; Peltier and Thiel, 1927).

From the extensive investigations carried out during the past 30–40 years in connection with the nature and development of cereal and grass rusts, it appears that in the case of these fungi—as with potato blight, *Phytophthora infestans*—there is, in addition to the vegetative mycelial stage, another vegetative stage in which the fungus exists in the cells of the host plant as a formless, plasmal body—a sort of plasmodium—symbiotically fused with the protoplasm of the cell, and to which the name mycoplasm has been given.

The mycoplasm-containing cell has an entirely normal appearance and contains nucleus, chlorophyll granules, etc. There is here no evidence that the fungus preys upon the host plant, the mycoplasm appearing to take the place completely of the protoplasm. There is ground for the assumption that in those cereals which are highly susceptible to the fungus, it is present in this condition in most of the chlorophyll-containing cells of the plant, even in those of the ears and

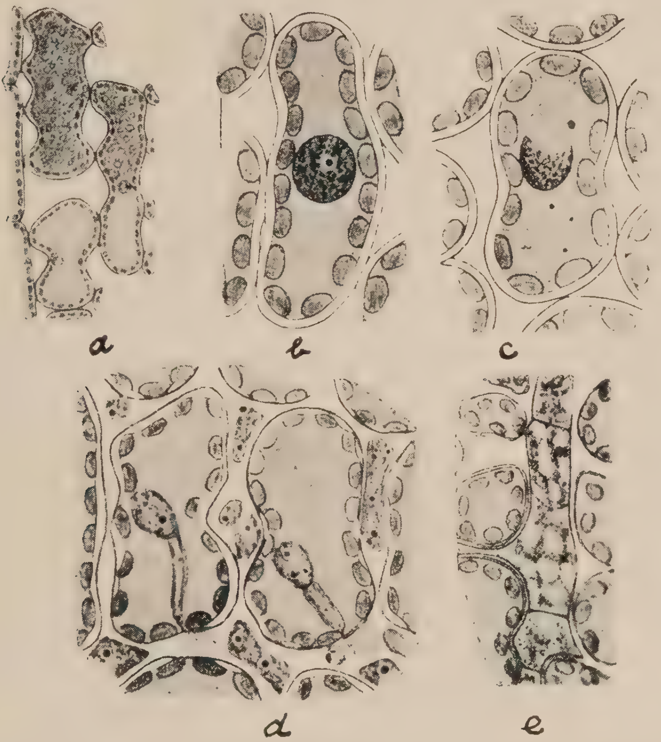

FIG. 107.—Mycoplasm and its change into Mycelium.

a, leaf cells of wheat seedling, 44 days after seed sown, containing mycoplasm in resting stage, cell nucleus and chlorophyll granules. *b*, leaf cell of plant of winter rye, 1–2 weeks before summer outbreak of brown rust; unhealthy enlargement of cell nucleus. *c*, leaf cell from the vicinity of the first uredo-pustule, mycoplasm in maturing stage; the cell nucleus in process of dissolution, small nucleoli appear in the plasm. *d*, leaf cells of an oat plant, from the vicinity of the first uredo-pustule; the mycoplasm pushes its way in a tubular form into the intercellular spaces to form mycelium. *e*, young mycelium with septa. (Orig.)

flowers. The time during which the fungus remains in this latent state varies, it may be 4–5 weeks, or as many months, perhaps even some years. This condition can therefore be regarded as the resting or dormant stage of the mycoplasm. Sooner or later, at a certain time of the year and under favourable external conditions (nature of soil, moisture, warmth, light, etc.), which may vary with the different rusts, a new stage in the life of the mycoplasm is entered upon—the maturing stage. The fungus frees itself from the symbiotic complex, penetrates the walls of the cell, and enters the adjoining intercellular space as a naked nucleoli-containing plasmodium which soon develops into a thin-walled branched mycelium and produces pustules. The maturing stage lasts only for a short time, at most a few days, perhaps only a few hours. Once the intercellular mycelium has begun to form, only a week is required for the rust pustules with spores to show themselves on the surface.*

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* The main features of the mycoplasm theory in its relation to the cereal rusts were brought forward in 1897 (Stockholm, Paris, Berlin). As the theory was opposed to the prevailing views on the subject and led to a modification of the methods of control of plant diseases it is easily understood that it met with opposition. The replies to the various criticisms will be found in the following publications : Ann. Sc. Nat., T. 15, 130–152, 1901 ; Ark. Bot., Bd. 1, 139–146, 1903 ; K. Sv. Vet. Ak. Handlg., Bd. 39, No. 5, 4–26, 1905 ; Ark. Bot., Bd. 5, No. 3, 39–54, 1905 ; Biol. Zentr.-Bl., Bd. 30, 618–623, 1910 ; Sitz.-Ber. Ak. Wien, Mat. Nat. Kl., Bd. 119, 1043–1050, 1910 ; Zentr.-Bl. Bakt. Abt. 2, Bd. 32, 453–459, 1912 ; Phytopath., Vol. 11, 385–388, 1921 ; and K. Sv. Vet. Ak. Handl., Bd. 62, No. 5, 170–186, 1921. A short résumé of the present position of the theory is given in the publication " La théorie du Mycoplasma, sa portée scientifique et sa perspective pratique," Inst. Intern. d'Agric., Bull. Rens. Agr. et Mat. Pl., Ann. 13, No. 3, 12, March, 1922.

Since 1903 a number of new cytological investigations have been made in connection with the development of the uredo-pustules of different cereal rusts (Allen, 1923 ; Kharbush, 1926 ; Buttle and Fraser, 1927 ; Allen, 1928). As, however, these new publications, like the older ones of Marshall Ward (Eriksson, 1903), do not refer to the primary pustules arising from an internal source of disease (mycoplasm) but only give information concerning the secondary pustules due to infection by uredospores already present, the results of these recent researches have no bearing on the mycoplasm theory, which can rightly, therefore, be retained.

Rusts of Cereals and Grasses

Puccinia

This genus contains a larger number of species than any of the other genera of the rust fungi. The winter spores are two-celled and contained in open, or, sometimes, closed, circular or elongated sori. In rare cases, one-celled winter spores are found intermingled with the two-celled spores.

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Black (Stem) Rust of Cereals and Grasses

Puccinia graminis Pers.

This fungus was the first of the many cereal rust fungi to attract attention, and it is one of the best known. It is found on rye, wheat, oats, and barley as well as on many grasses. In Sweden the disease generally appears about the middle or end of July, and on autumn-sown corn some weeks earlier than on spring-sown. The symptoms of the disease are elongated, reddish-brown pustules of varying size and filled with spore dust. These pustules, which are scattered over the leaves and stems, and are found most abundantly on the leaf sheath, often also on the ears and panicles, form the uredo-stage of the fungus. The uredospores are elliptical and covered with spines, and have generally four equatorial germ pores. In a moist environment, rain or dew, the uredospores generally germinate within a few hours. If germination takes place on a suitable substratum—for example, spores from oats germinating on a young oat leaf—the germ-tube enters the leaf and produces a group of new pustules, filled with the same spore dust, after 8–10 days. Further generations follow one another as long as young, green shoots are present, perhaps for 2–3 months.

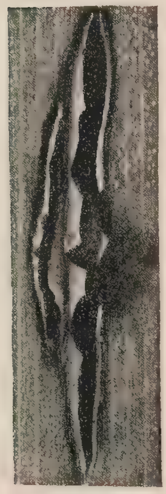
FIG. 108.—Uredo-pustule of *Puccinia graminis* on Rye. (Orig.)

Up to the end of the last century it was thought that spores from one cereal could carry the disease to another species, and that infection could spread from a cereal to those grasses on which black rust was found, and conversely that the grasses could infect cereals. This view is, however, incorrect; there are several specialised forms of the black rust fungus, and these are more or less fixed on one or more host plants.

This specialisation on the part of one and the same species of fungus has not, however, been identical in different countries. In Sweden the following specialised forms have been distinguished :

1. f. sp. Secalis on rye and barley and 9 species of grasses ;
2. f. sp. Tritici on wheat and in rare cases on rye, barley, and oats ;
3. f. sp. Avenæ on oats and 21 species of grasses ;
4. f. sp. Airæ on *Aira cæspitosa* and *A. bottnica ;*
5. f. sp. Agrostis on *Agrostis canina*, *A. vulgaris*, and *A. stolonifera ;*
6. f. sp. Poæ on *Poa compressa*, *P. cæsia*, *P. serotina*, and *P. pratensis ;*
7. f. sp. Epigæi on *Calamagrostis Epigeios* and *C. varia ;*

? 8. f. sp. Festucæ on *Festuca pratensis ;*
? 9. f. sp. Hierochloæ on *Hierochloa borealis ;* and
? 10. f. sp. Holci on *Holcus lanatus*.

In Russia (v. Jaczewski, 1910 ; Teterevnikova, 1927) the following forms have been distinguished :

1. f. Secalis on rye, in rare cases on barley and on 6 species of grasses ;
2. f. Tritici on wheat and barley and 4 species of grasses ;
3. f. Avenæ on oats and 5 species of grasses ;
4. f. Airæ on *Aira cæspitosa ;*
5. f. Agrostis on *Agrostis alba*, in rare cases on rye, wheat, barley, and oats and on 6 species of grasses ;
6. f. Poæ on *Poa compressa*, *P. pratensis*, and *P. serotina ;*
7. f. Calamagrostis on *Calamagrostis Epigeios ;*

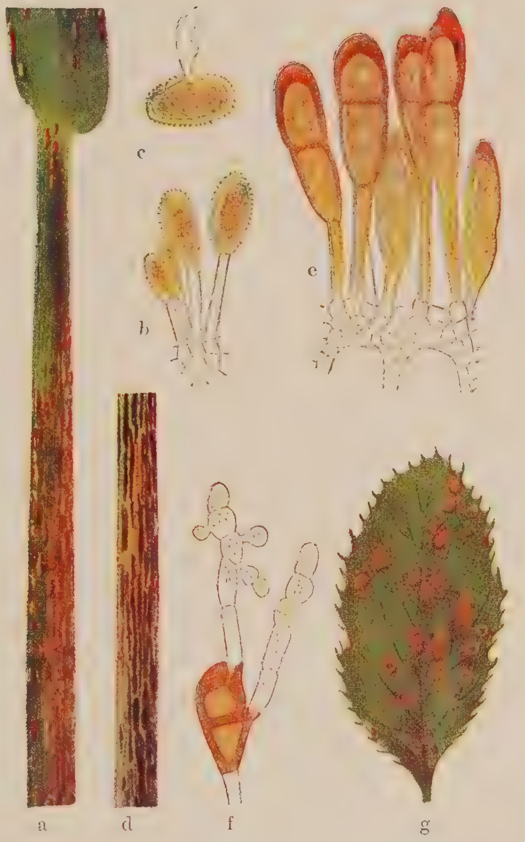

FIG. 109.—Black Rust of Oats.

a, stem of oat with uredo-pustules ; *b* and *c*, summer spores, one of which is germinating ; *d*, stem of oat with puccinia-pustules ; *e* and *f*, winter spores, one of which is germinating ; *g*, barberry leaf with æcidia. (Orig.)

[*To face p.* 150.

8. f. Aperæ on *Apera spica-venti*; and
9. f. Arrhenateri on *Arrhenatherum elatius* (and on oats).

In North America the specialisation is carried out in a different manner (Carleton, 1890; Freeman and Johnson, 1911; Stakman, Levine, and Bailey, 1923; Stakman and Levine, 1924; Bailey, 1925; Levine, 1928).

1. f. Secalis on rye;
2. f. Tritici on wheat and barley and 9 species of grasses;
3. f. Hordei on barley and wheat, in rare cases on oats and rye;
4. f. Avenæ on oats and 9 species of grasses;
5. f. Poæ on *Poa compressa*, *P. annua*, *P. fertilis*, *P. nemoralis*, and *P. triflora*.

The specialisation of cereal rusts has been closely studied in America and by means of extensive inoculation trials, generally with the uredo-stage of the fungus on various cereals and grasses, it has been possible to differentiate biologic strains within one and the same specialised form of fungus. These strains, which may be regarded as similar to the so-called "small-species" found in genera such as *Hieracium*, *Taraxacum*, *Rosa*, *Draba*, *Euphrasia*, etc., show their biological differences in the following manner: Each strain has a different power or capacity of infection in regard to individual varieties and stocks of cereals and grasses coming from different districts. This power may be considerable or only small, or it may be entirely absent. Further, it has been possible by microscopical measurements of many hundreds of spores, in some cases uredospores and in others teleutospores, to observe morphological differences in the size and shape of the spores of different strains.

The American research stations have paid special attention to the fungus of black rust, f. sp. Tritici, in this connection. In 1917 one form—f. sp. Tritici-compacti—was distinguished which infected many varieties of autumn-sown wheat, but failed to infect a number of spring-sown varieties. Similar discoveries were made later on, and by October, 1918, the number of separate strains had risen to about a dozen, and in 1922 to 37 in the United States. These strains are distinguished by Roman numerals I to XXXVII. No single one of the 37 strains of the fungus has been able to infect all the 130 or so varieties of autumn- and spring-sown wheat, and no species or variety of Triticum (with the exception of a single variety, *Triticum dicoccum*) has shown itself resistant to all the strains of the fungus. The biological, as well as the morphological, characters of the individual strains have been found, after many years' investigation, to be constant and to be independent of temperature, light, climate, and other local conditions. The distribution of these strains is still uncertain, but it is considered that about half of the number are found in each wheat-growing district in North America. In Western Canada about 14 such strains have been recorded (Newton, 1922).

The examination in 1923 of more than 100 samples of rusted oats from 24 States in America, and infection tests made with some 70 different species, varieties, and stocks of the genus *Avena*, led to 4, probably 5, different strains of f. sp. Avenæ being distinguished, all of which showed constant characters.

It is clear that the rust problem is greatly complicated by these discoveries, as new and biologically different forms arise owing to specific local conditions (Melchers and Parker, 1922; Blaringhem, 1923). Proof has, indeed, been given that when two different stocks of wheat react differently to two different strains of the fungus, in that one stock is resistant to one strain and the other stock to the other strain, it is possible by crossing these stocks to produce a new wheat that is resistant to both strains of the fungus. Having regard to the many different strains of the fungus and the birth of new ones, and seeing that the value of a variety of wheat does not depend solely upon its resistance to rust, as the numerous breeding trials carried out in America with different rust-susceptible varieties and lines have shown, both in the case of wheat (Hayes, Stakman, and Aamodt, 1925; Kiesselbach and Peltier, 1926) and oats (Hayes, Stevenson, and Lunden, 1928), but upon many other characters such as resistance to other pests, time of ripening, yield, resistance to frost, milling properties of the grain, etc., it is clear that in spite of the investigations to which reference is made above, *the question of cereal rusts is even now far from being cleared up conclusively.*

About two weeks after the first appearance of the uredo-stage of the fungus the teleuto-stage appears. In this stage there are elongated lesions which often coalesce and are filled with a black, powdery mass, hence the name "Black Rust." The spores are two-celled, thick-walled, and they taper at the apex. After a natural hibernation in the open, these spores are able to germinate in the following spring. If, however, the spores have been kept over the winter in a room—either warm or cool—or if they have lain in the corn stack, they cannot be brought to germination in the spring. Thus rusted straw stored in the barn or stack is not likely to infect the next year's crop. Naturally hibernated spores germinate best in April and May, and less well in summer; by the autumn the power to germinate has, as a rule, been lost.

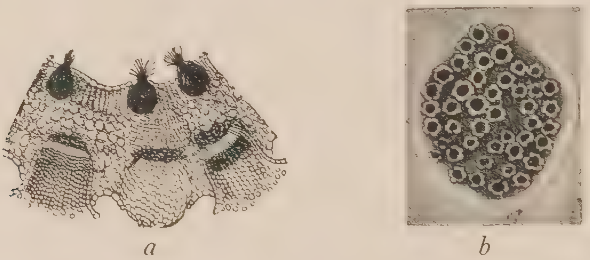

Fig. 110.—Barberry Rust.
a, Barberry leaf, pycnidia above, æcidia below; *b*, patch of rust from under surface of leaf. (*b*, Orig.)

The viability of the spores is not reduced if they are ploughed under with the stubble, and it has been shown that pieces of rusted straw, buried to depths of 8–20 inches in October, germinated their spores successfully when dug up on May 1 of the following year.

The over-wintered spores germinate when placed in a warm, moist atmosphere. Each of the two cells sends out a germ-tube, a basidium or promycelium, which is divided into several segments by transverse walls, and from each segment (usually four) a basidiospore is abstricted.

When the basidiospores (sporidia) germinate on a young leaf, shoot, or ovary of the barberry shrub (*Berberis vulgaris*), or on the young ovary of the cultivated garden shrub *Mahonia Aquifolium*, the barberry rust or cluster cup—*Æcidium Berberidis*—is produced. The rust patches on the barberry leaves are almost circular; on the upper surface of the leaf they are red in colour with numerous small, black dots which are the mouths of the flask-shaped pycnidia embedded in the leaf. On the lower surface of the leaf are found large, yellow patches with numerous, open cup-like æcidia. In the pycnidia, very small rod-like pycnoconidia develop. The æcidia have a ragged, reflexed margin, and they contain numerous æcidiospores which arise in beadlike rows from the bottom of the cup.

This æcidial stage generally appears in Sweden during the latter half of June. The spores germinate irregularly; the alternation of cool nights and warm days is favourable to germination.

If æcidiospores from a cluster cup which has been formed as the result of infection from a rusted oat stem come to germinate on a young oat plant a group of uredo-sori generally appears in 8–10 days. A similar state also arises when the æcidiospores germinate on other species of grasses which are carriers of the same biologic form of rust as oats (f. sp. *Avenæ*). If, on the contrary, the form mentioned above germinates on rye, barley, etc., no uredo-pustules are produced. In the same way æcidiospores, which have arisen on barberry as a result of infection by rust spores from rye or barley, produce uredo-pustules on rye and barley but not on wheat or oats.

The biological differences in the various forms of the barberry rust have only been understood since the end of the last century, and they naturally play an important part in regard to the influence of barberry in spreading black rust in cereal cultivation. If, for example, barberry is infected through rusted oats, *Dactylis glomerata*, *Alopecurus pratensis*, etc., the rust can be spread to an oat-field but not to fields of rye, wheat, or barley. On the other hand, a barberry bush infected by rust from rye, barley, *Triticum repens*, etc., can spread rust to rye and barley, but not to wheat or oats. In this way the part played by barberry in connection with the ravages of black rust is considerably limited.

The question of the real importance of barberry in the spread of the black rust of cereals still remains the subject of discussion, both from the practical and the scientific standpoint. On the one hand, in Denmark (Kölpin-Ravn, 1918) and the United States (Davis, 1907; Kempton, 1921; Stakman, 1923), the barberry stage is regarded as an essential link in the development of the fungus—at least in countries with a cold climate. On this account, it is held that the extermination of every barberry bush is a certain means of getting rid of rust. On the other hand, the view is held that the barberry stage is only facultative and that the fungus can continue to live without passing through it. The extermination of barberry, which is difficult and expensive to carry out, is, therefore, of very doubtful value.

This latter view is mainly based on the following considerations: (1) A study of the older literature leads to the opinion that the uredo- and puccinia-stages of the disease on cereals are much older (reckoning in centuries) than the æcidial stage on the barberry (Eriksson, 1918). (2) The spread of the fungus from an infected barberry bush has not been traced to a greater distance than 50 metres (Eriksson, 1896). (3) Black rust is found, and causes heavy losses, in all corn-growing countries of the world, even where, as in India, Australia, and Uruguay, barberry does not exist (McAlpine, 1906; Eriksson, 1918; Waterhouse, 1923). (4) Experiments made to ascertain whether in the absence of barberry the disease over-winters in the uredo-stage or whether spores are transported in the upper air have not yielded any conclusive results. (5) The specialisation of the fungus and particularly the division of the original specialised form into numerous biologically distinct strains, greatly reduces the importance of barberry as a danger to cereal growing. (6) The different times at which the main outbreaks occur—early in summer, when barberry is present, and 1–1½ months later when it is absent—as well as the sudden appearance of uredo-pustules over the whole field, leads to the conclusion that in addition to external infection of the cereal plants there must be some form of internal infection which is responsible for the later outbreak (Eriksson, 1900, etc.). (7) The symbiosis between the protoplasm of the plant and that of the fungus, which has been termed *Mycoplasm*, has been demonstrated as well as the passage, step by step, of the fungous element from the plasm stage into an independent mycelium stage (Eriksson, 1900, etc.). These observations furnish a natural and complete explanation of the problem of hibernation.*

It follows that if barberry rust is an accidental and unnecessary stage in the development of the fungus the extermination of barberry bushes in the vicinity of corn-fields would involve expenditure and be of secondary value.

With a view to proving experimentally the real nature of the heterœcism of the fungus—obligatory or facultative—an energetic war of eradication has recently been declared against the barberry bush in America. The number of eradicated bushes amounted in Illinois (1918) to 328,000, in Colorado (1918–19) to 35,000 (on 1,150,000 acres), in Michigan to 458,783, in Montana (1918–25) to 11,100, altogether in thirteen States (1918–26) to 14,000,000 bushes. In Canada (Manitoba) the number of bushes eradicated (1917) was 3,000.

This radical destruction of barberry bushes over large areas has not, however, completely prevented the appearance of black rust in the cornfields. The first appearance of the disease was delayed by about three weeks, and consequently there was a reduction in the annual loss of crop. In North Dakota the reduction was estimated to be from 17,800,000 bushels (1915–24) to 1,780,000 bushels (1925), and in thirteen states together, from 50,419,666 bushels (1915–20) to 15,920,000 bushels (1921–26); the losses still amount, it will be seen, to an important figure.

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* This brief account of the true importance of barberry in relation to the growing of cereals is further developed in Landw. Vers. Stat., 1897, 83–95; in K. Vet. Ak. Handl., Bd. 39, No. 5, 1905, 4–23; in Ill. Landw. Ztg. Jahrg. 27, 1907, No. 41, 22, May, in Zentr. Bl. Bakt., Abt. 2, Bd. 48, 1918, 395–400; and Tidskr. Landtm., 1916, 793–798 and 816–819. Compare, also, Ann. Sc. Nat. Paris, Sér. 8, T. 14, 1900, 119–124 (Puccinia sessilis on Phalaris arundinacea).

In the light of the mycoplasm theory the retarded appearance of the uredopustules is easily understandable, as it can be referred to the change of the fungus from the internal, latent plasm stage within the host plant to the mycelium stage. A similar retardation in the appearance of the uredopustules also occurs in cases where the disease breaks out at long distances from barberry bushes. Those who do not accept the theory have no alternative but to ascribe the source of the disease to a distant barberry bush, perhaps many miles away, from which the disease is either carried directly by the wind or by intermediate uredo-centres serving as bridges.

Recently some detailed accounts have been given of the appearance of the different stages of the disease in America. In Colorado, during five years' observations, barberries showed infection as early as May 8, and wheat by June 1. Along the eastern border of the State the earliest appearance of rust during the past seven years, in the absence of barberries, has been June 22–25, on which date infection could be found only 5–10 miles west of the State line. In the irrigated sections of Colorado, some 250 miles west of the eastern border, infection occurs 12–17 days later than the first attacks in the eastern part of the State, in the absence of local infection from barberries (Durrel and Lungren, 1927).

In the meantime it is found, both in Europe and America, that the statements of experts tend to disclose a certain doubt as to the obligatory heterœcism of the fungus. Thus from Italy: It is regarded as not yet certain whether the absence of the æcidial host interferes with the prolonged propagation of the fungus (Petri, 1926). From France: What is not yet clear is whether the absence of the æcidial host may interfere with the prolonged propagation of the fungus (Ducomet, 1926). It is reported from Western Canada that no distinct connection can be traced between the occurrence of rust on barberries and the outbreak of local or general epidemics in Manitoba, Saskatchewan, and Alberta (Jackson, Fraser, and Bailey, 1925).

There is no doubt, however, that with this fungus, as with other cereal rusts, external factors such as situation, drainage, chemical and physical properties of the soil, manuring, preceding crop, time of sowing, climate, etc., are connected with the severity of the outbreaks of disease. Although many experiments have been made in this connection the results are not sufficiently definite to admit of practical conclusions being drawn from them.

For control measures, see below, p. 173.

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Puccinia Phlei-pratensis Eriks. & Hen., Timothy Grass Rust, causes brown uredo-sori of varying size on the leaves, leaf sheaths, and stems of *Phleum pratense* and *Festuca elatior*. These sori are very similar to those of black rust but slightly smaller. Sometimes black patches of teleuto-sori, also similar to those of black rust, are found on the stems, but they are much less extensive. Attempts have been made to transmit this rust to the barberry but without result, except possibly in one or two cases. The sori of the true black rust (*Puccinia graminis* f. sp. *Avenæ*) have by chance been observed on *Phleum Böhmeri*, *Ph. Michelii*, and *Ph. asperum* (Eriksson, 1902).

In America, where this disease is mentioned in the literature, it has wrought havoc in the timothy fields (Maine, Ontario, Minnesota, Iowa, Kentucky, and Virginia). Although by inoculation trials it has been more frequently transmitted in that country than in Europe to other species (*Avena sativa*, *Secale cereale*, *Festuca elatior*, *Dactylis glomerata*, *Arrhenaterium elatius*, and *Poa compressa*), its appearance and distribution in the open have apparently nothing to do with the forms of black rust which occur on the plants mentioned (Kern, 1909 ; Johnson, 1911 ; Mercer, 1911).

In St. Paul (Minnesota), considerable differences in rust resistance, ranging from complete susceptibility to very high resistance, were observed in experiments carried out from 1918 to 1922 and in 1925 with various self-fertilized lines of timothy grass, but no definite evidence of biologic specialization of the fungus was detected (Barker and Hayes, 1924 ; Proytchoff, 1928).

Brown Rust of Rye

Puccinia dispersa Eriks.

This rust is found only on rye. The uredo-sori appear about the middle of June on autumn-sown rye and a few weeks later on the spring-sown varieties. Sometimes a few solitary uredo-pustules may be found on autumn-sown rye about a month after sowing or early in spring, a

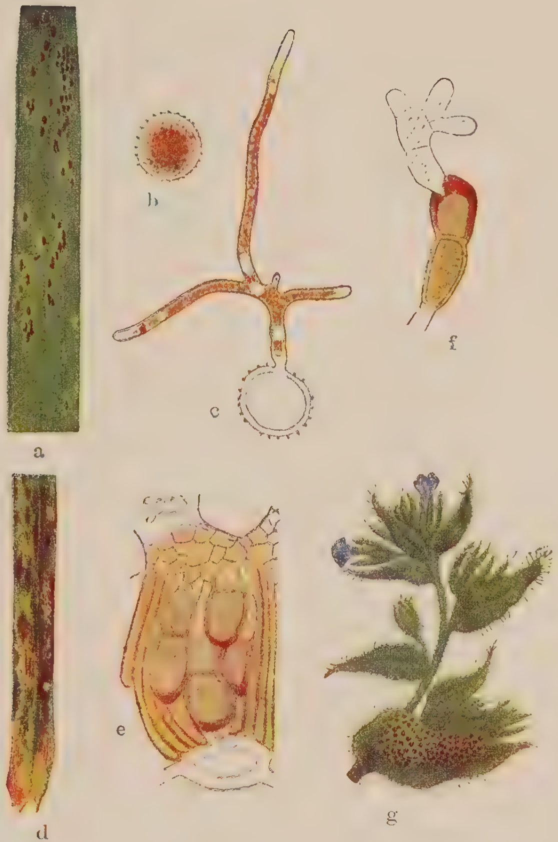

FIG. 111.—Brown Rust of Rye.

a, leaf of rye with uredo-pustules; *b* and *c*, summer spores, one of which is germinating; *d*, leaf of rye with puccinia-patches; *e*, collection of winter spores; *f*, germinating winter spore; *g*, leaf of Anchusa with æcidia. (Orig.)

few weeks after the snow has gone. This early (proleptic) outbreak terminates sooner or later of its own accord, and is not connected with the main outbreak in summer.

The uredo-pustules appear in the form of chocolate-brown, irregularly scattered spots on the leaf blade, chiefly on the upper surface. The spores are spherical and as a rule they germinate easily. A few weeks after the appearance of the uredo-pustules the black groups of teleutospores, covered by the epidermis, can be seen on the under surface of the leaf. The teleutospores, in the case of this variety, are able to germinate in the same autumn, as soon as they have developed fully and are ripe.

In America (Indiana) collections of teleutospores from eleven States were made in the spring and summer of 1919 and were tested for germination from July 15 to April 7, 1920, and over-wintered out of doors. The teleutospores were found to be capable of germination whenever conditions were favourable, either in the autumn or, surviving the winter, in the spring, or both. Varying climatic conditions in different years may tend to level such a distinction.

Should germination take place on the young leaves or other parts of one of the alkanets (*Anchusa arvensis* or *A. officinalis*) a cluster cup rust (*Æcidium Anchusæ*) is formed. This æcidial stage is found, in the case of southern Sweden, as early as August and September on species of *Anchusa* growing there as weeds. In central and northern Sweden this form of rust is rare and the plants just mentioned are usually not affected, although rye crops in the vicinity may be badly attacked by brown rust.

The conditions appear to be the same in other countries. In France (Grignon), where species of *Anchusa* are commonly found on rye-fields, it is often impossible to find a single æcidial pustule. Thus in 1925, in spite of the most diligent search prior to the rye being harvested, not one æcidial pustule could be discovered (Ducomet, 1926). In Switzerland (Bern), *Anchusa arvensis* appears sporadically and only rarely bears æcidia (Fischer, 1927). From America (Indiana) it is reported that it is not probable that the æcidial hosts of *Puccinia dispersa* are of much importance in that country, and æcidia have never been collected in North America on *Anchusa arvensis* (Mains and Jackson, 1924).

The æcidiospores germinate easily and quickly ; if young rye plants are growing in the vicinity of rusted weeds they are soon infected and uredo-pustules appear on them in 8–10 days. When cold weather begins in winter the above-ground parts of alkanet plants die down—in the case of *Anchusa arvensis* the whole plant dies—and only the seeds remain alive. In this way the æcidial stage of the fungus is destroyed, and so the fungus is not able to cause a fresh outbreak of the disease on rye in the following year. Further, there is no over-wintering of the teleutospores as they germinate in the autumn. Finally, as no hibernating uredo-stage has been detected, either in the form of spores or mycelium, it must be assumed that this fungus over-winters solely as a vegetative plasm.

Observations made in Switzerland in the winters of 1925–26 (Jan. 6, Feb. 6, April 2, June 6) and 1926–27 (Feb. 28) with respect to the appearance of a few scattered uredo-pustules, are considered to prove that (in the vicinity of Bern) the fungus is able to pass the whole annual cycle in the uredo-stage. These observations are not, however, free of certain criticism as regards rust-free bridging periods : in 1926, on one rye-field from April 2 to May 2, and on all the fields from August 2 to the middle or end of September, *i.e.* 1½–2 months ; in 1927, from February 28 to the time of the main outbreak in summer (Fischer, 1927).

As to the correctness of these conclusions, an opinion must be reserved as long as no week-by-week systematic investigations are presented, whereby a direct genetic connection between the sporadic appearance of the uredo-stage in the winter months on the one hand and the mass outbreaks, which occur almost simultaneously in the following summer, is finally proved. The few winter uredo-pustules met with from time to time must be regarded as accidental remnants, of the previous year's uredo-generation, due to local weather conditions and of no functional importance in reference to the reappearance of the epidemic in the next year.

This form of rust is of great economic importance in the United States as in some years it causes a serious reduction in the crop yields, for example, in 1910 by 538,000 bushels. Trials which were undertaken with sixty different varieties of rye to test their resistance to the fungus showed that in no case was there immunity, but that individual members of many varieties possessed considerable resistance, almost to the point of immunity.

For control measures, see below, p. 173.

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Brown Rust of Wheat

Puccinia triticina Eriks.

This rust only attacks wheat. In Sweden it usually appears in the first or second week of June on autumn-sown wheat, and two or three weeks later on spring-sown varieties. It produces small, scattered, chocolate-brown uredo-lesions on the leaves, mainly on the upper surface. The spores are spherical and germinate irregularly, often badly. A few weeks after the uredo-pustules appear, the winter stage of the fungus is produced in the form of black sori arranged in rows on the under surface of the leaf, in severe attacks also on the leaf sheaths; the sori are covered by the epidermis of the organ on which they are present. The spores, which are similar in appearance to those of the brown rust of rye, hibernate and then germinate in the following spring.

The reaction of different kinds of wheat seems to be in some degree unequal. Trials carried out in America (Indiana) in 1918 led to at least twelve biologic forms of the fungus being distinguished by the manner in which they infected eleven selected, different strains of wheat. All these varieties were found to be more or less resistant to one or more forms of the fungus. The biologic forms are generally found to be mixed in the field (Mains & Jackson, 1926).

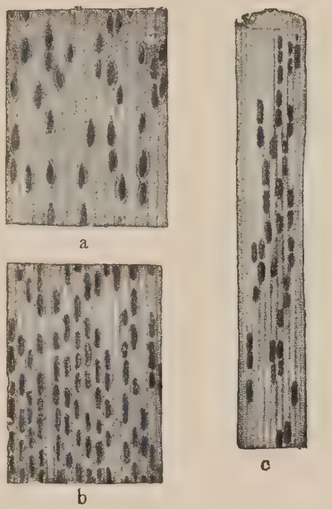

FIG. 112.—Brown Rust of Wheat.

a, leaf of wheat and uredo-pustules; *b*, leaf; *c*, stem of wheat with puccinia-patches. (Orig.)

In Germany, in 1925, a series of experiments was undertaken in the inoculation of about 140 species and varieties of winter and summer wheat with spores of the fungus. Of the 88 varieties of winter wheat, about 23 showed heavy infection; of the 52 summer wheats 32 were very susceptible. None of the varieties tested was completely immune; *Triticum monoccum*, however, proved highly resistant (Rudorf, 1927).

In connection with the American variety trials just mentioned, extensive experiments have been conducted since 1920 in certain parts of the United States (in the field at Washington, Knoxville, and La Fayette, and in the greenhouse at La Fayette and Manhattan), with a view to studying the inheritance of resistance to this rust of crosses between resistant and susceptible varieties of common wheat. The trials were carried to the F^4 generation, but the results were not such as to hold out much promise for practice.

Somewhat similar results were obtained from crossing experiments carried out in Japan with the object of exploring the correlations between morphological character, chromosome number, and resistance to the fungus (Tochinai and Kihara, 1927).

In America (La Fayette) the spermogonium stage was produced in

1920 on *Thalictrum Delavayi* and *Th. flavum*, also in Russia (Leningrad) on *Thalictrum minus* (Eremeyeva, 1925). In France (Clermont-Ferrand) æcidiospores from *Thalictrum minus* gave rise to visible uredo-pustules on *Triticum vulgare* after 11–12 days (Alabouvette and Meneret, 1927).

For control measures, see below, p. 172.

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Puccinia bromina Eriks. forms brown, later black, lesions on leaves, stems, and glumes of various brome grasses such as *Bromus arvensis*, *B. asper*, *B. secalinus*, *B. racemosus*, *B. mollis*, *B. sterilis*, *B. tectorum*, etc., but, as far as is known, not upon *B. erectus* and *B. inermis* in Sweden. On the two last-mentioned species of *Bromus*, a similar but heterœcious species **Puccinia Symphyti-Bromorum** F. Müll., which gives rise to an æcidial stage on *Symphytum officinale* and *Pulmonaria montana*, has been observed in more southerly countries, such as Switzerland. The above two rusts are often stated to be identical, but whether this view is correct has not yet been decided.

Puccinia holcina Eriks. forms reddish-yellow lesions on the leaves, and afterwards black, discontinuous streaks on the leaf sheaths of Yorkshire fog (*Holcus lanatus*) and creeping soft grass (*Holcus mollis*).

Puccinia Triseti Eriks. produces small, bright yellow pustules which are distributed over the upper surface of the leaves of yellow oat grass (*Trisetum flavescens*). Later, black spots appear on the under surface of the leaves.

Yellow Rust of Wheat, etc.

Puccinia glumarum (Schm.) Eriks. & Hen.

This rust may be placed along with black rust in respect of the damage which it causes. It attacks wheat, barley, rye, and various grasses.

In Northern Europe, yellow rust appears chiefly on wheat, especially autumn-sown wheat, the main outbreak occurring usually at the middle or end of June. The fungus makes its appearance about the same time

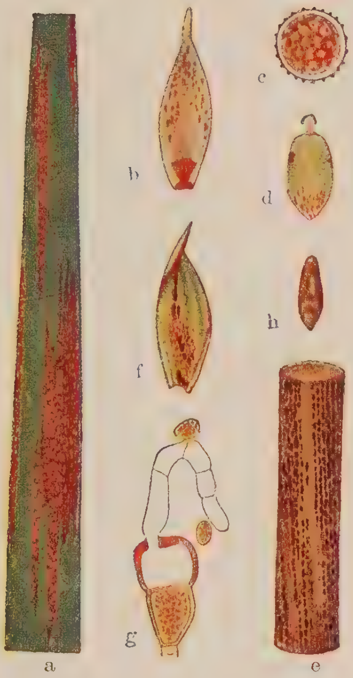

Fig. 113.—Yellow Rust of Wheat.

a and *b*, leaf and glume with uredo-pustules; *c*, summer spore; *d*, young grain of wheat with uredo-pustules under skin; *e*, stem, and *f*, glume with puccinia masses; *g*, germinating winter spore; *h*, shrivelled, rusted grain. (Orig.)

and to about the same extent on each plant in the field. The attack usually occurs when the plants are about 3 feet high, generally in one of the middle leaves which otherwise is growing strongly and is of a deep green colour. The fungus gives rise to small, lemon-yellow uredo-pustules arranged in long streaks on the upper surface, and in a week or two the greater part of the surface of the leaf is covered with these pustules. In years when the attack is severe the fungus also appears on the ears, on the inner side of the glumes, or even upon the skin of the young grains. The spores are spherical and germinate capriciously.

A few weeks after the first uredo-pustules occur the teleuto-stage appears, first of all on the leaves that are already infected and on the leaf sheaths. These teleuto-sori form long streaks of minute, brown, later black, spots mainly on the leaf sheaths, and they are covered by the epidermis. In severe cases the spots appear on the inner side of the glumes and in the wall of the grain. Such grains shrivel up when ripe and are known as "rust grains"; they do not, however, play any part in the economy of the fungus.

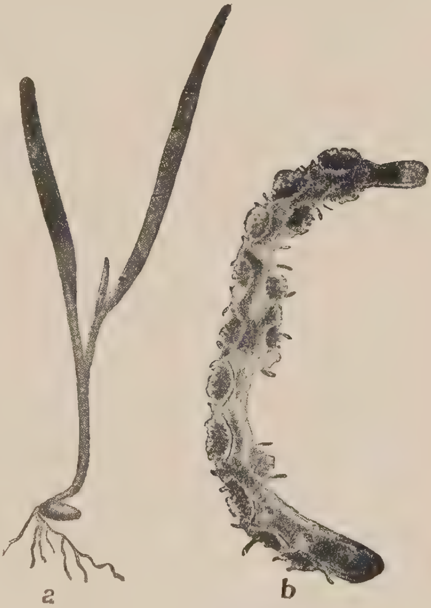

FIG. 114.—Yellow Rust of Wheat. *a*, young wheat plant with uredo-pustules on the first leaf (proleptic outbreak); *b*, rusted leaf of seedling in section. (Orig.)

The teleutospores germinate in late autumn at the time when wheat is sown. Probably the fungus enters the seedling and after passing a period, which may vary from one to several months, in the latent plasm form breaks out as open uredo-pustules on the leaves and stems of the developed plant. It is true that no anatomical or experimental proof has been obtained for this assumption, but numerous field observations and the lavish production of teleutospores, which otherwise would be of no account, favour the view.

All the inoculation experiments so far made with the teleutospores of this fungus have yielded negative results both on grasses and other plants, so probably no æcidial stage occurs.

Not infrequently outbreaks of disease, of varying virulence, appear during late autumn or in very early spring on the seedlings of autumn-sown wheat. Neither of these outbreaks stands in direct relation to the main outbreak which occurs from the middle to the end of June. The autumn outbreak is interrupted by the winter, and the spring outbreak is separated from the normal summer outbreak by a shorter or longer period in which no rust occurs. These premature (proleptic) outbreaks are comparable with the not uncommon instances of plants which normally flower in the spring, but under special climatic conditions produce some flowers in the preceding autumn. Severe proleptic outbreaks of disease are not to be regarded as indicators of a bad rust year, as this is largely determined by the weather conditions in May and June.

In Europe, where this fungus has been known in the literature for more than a hundred years (1815) under the collective name *Puccinia Rubigo vera* DC., the following specialised forms are distinguished: (1) f. sp. Tritici on *Triticum vulgare ;* (2) f. sp. Secalis on *Secale cereale ;* (3) f. sp. Hordei on *Hordeum vulgare ;* (4) f. sp. Elymi on *Elymus arenarius ;* and (5) f. sp. Agropyri on *Agropyrum repens.* In addition, the fungus has been recorded on 9 other species of *Triticum* and on 2 other species of *Hordeum*, as well as on 2 species of *Ægilops*, 1 of *Dactylis*, and 1 of *Calamagrostis.*

The chief host plant, at least in Northern Europe, is wheat, but this variety of rust sometimes appears on rye with serious results ; in 1904 in Austria 30 per cent. of the autumn-sown rye was diseased and 75 per cent. of the spring-sown (Köck, 1904).

In the western parts of North America, where the yellow rust was first observed in 1915 on the Pacific Coast and in the Rocky Mountains district (Kölpin-Ravn), the fungus appears on wheat, barley, rye, spelt, and emmer, as well as on 33 species of wild grasses. The fungus has been transmitted to 26 other species by inoculation. Altogether 59 species of wild grasses in the United States are known to act as hosts to yellow rust. The wheat form, f. sp. Tritici, can pass to rye (in certain cases) and to barley (in rare cases) as well as to 47 wild grasses amongst which are 19 species of *Bromus*, 11 of *Agropyrum*, 7 of *Hordeum*, 7 of *Elymus*, 1 of *Hystrix*, 1 of *Phalaris*, and 1 of *Sitanion.* In addition, there are 12 species of grasses upon which yellow rust has been found in the field, but it has not yet been determined whether these forms are identical with the yellow rust of wheat. There is reason to think that there are two or more different strains of f. sp. Tritici (Hungerford and Owens, 1923) in North America, where also f. sp. Hordei has been found.

The different varieties of any one cereal vary in their susceptibility to the yellow rust fungus. This applies particularly to autumn-sown wheat where a distinction can be made between (*a*) very susceptible ; (*b*) less susceptible ; and (*c*) almost non-susceptible. To the first of these groups belongs a relatively small number of European wheats and some American varieties that have been tested in Northern Europe.

To the second group belong most of the sorts which only suffer slightly from the disease in normal years but are seriously affected in a " yellow rust year " ; the majority of these wheats have been grown for a long time past. The third group, which includes varieties of *Triticum durum*, *T. turgidum*, and *T. monococcum*, hardly suffers at all from this disease.

In Finland (Helsingfors), extensive experiments were undertaken from 1921 to 1925 with 227 varieties and lines of spring wheat. The results showed marked varietal differences in reaction to this rust, due to internal hereditary factors. The degrees of infection being expressed by numbers—1 (most severe attack) to 10 (entirely rust-free)—the average resistance of 15 early and medium-early varieties was 3, compared with 5·7 and 8·7 for 50 medium-late and 80 late varieties respectively. It is thus seen that there is a distinct correlation between resistance to rust and delayed maturity.

Nine different crosses showed that when a resistant parent is used for crossing with varieties having different grades of resistance, the greater the degree of resistance of the latter, the greater will be the resistance of the F^3 progeny plots (Persola, 1927).

Experience has taught that the resistance of a variety of wheat, even the most susceptible, varies greatly in the same place in different years, and occurs especially when exceptional years succeed one another and the seed used comes from the harvest of the previous year. This was shown on Swedish trial plots in the years 1890–93. The year 1890 was a yellow rust year, 1891 almost free of the disease, 1892 again a rust year, 1893 an almost rust-free year. The differences in weather, time of sowing, etc., did not satisfactorily explain the wide variations observed during the above period of four years.

It has been thought that the greater or less degree of resistance to disease shown by a variety of wheat is a constant and inherent property of that variety. If, then, a wheat showing a high degree of resistance to disease but having drawbacks such as liability to die out in winter, poor yielding power, etc., were crossed with a variety in which the characteristics, other than resistance, were satisfactory, it should be possible to obtain a resistant wheat of high quality. It was even considered that the hybridization trials which have been in progress for the past twenty years (Biffen, 1907 ; Nilson-Ehle, 1909, 1911) had reached a point that assured the mastery over yellow rust. Unfortunately, recent experiences have shown this hope to be deceptive.

If, in any year, the combined climatic conditions (temperature, moisture, sunlight, wind, atmospheric pressure, etc.) are very favourable to the development of the yellow rust fungus, it is noticed that a wheat which for a number of years has shown itself to be resistant may be suddenly and disastrously attacked. An instance of this kind was seen in 1919 in the south of Sweden (Scania), a district in which wheat cultivation is at a high general level and the best varieties are grown. Most of the fields of winter wheat suffered severely from yellow rust. whereas in 1917 and 1918 not a trace of the disease could be found (Eriksson, 1922). Another similar case occurred in the same district in 1923, the very resistant wheat Pansar II being heavily rusted, whilst Pansar III and other varieties were completely immune (Nilson-Ehle, 1923).

The above facts lead to the conclusion that breeding experiments have not solved the problem of yellow rust on cereals, and that the question in general should not be regarded as an item in the programme of plant improvement. Mendelian laws and methods of work are valuable for the improvement of plants, but they are inadequate for the investigation of the life-history and activities of the parasite. An especial difficulty is introduced by the uncertain appearance of yellow rust from year to year; this makes an investigation on Mendelian lines difficult to carry out. The phenomena of disease must have a special study if the efforts to combat the disease are to succeed. Willingly or unwillingly, the plant breeder, if he is to be successful in dealing with this problem, must take into account the possible existence of an internal source of disease. As long as the eventuality of an internal source of infection being present in the seed corn or in the over-wintering parts of the stem is denied, the desired end will not be reached.

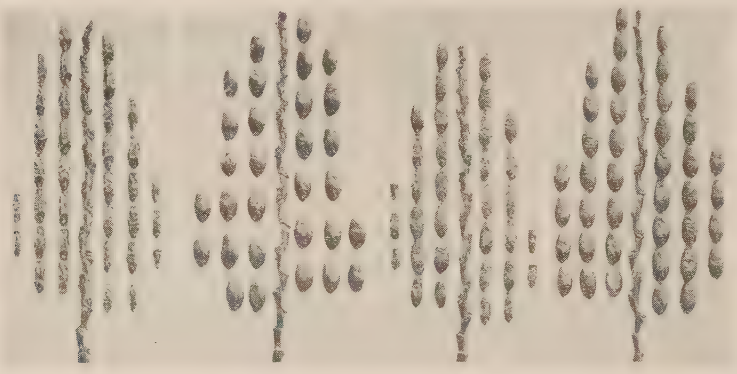

FIG. 115.—Ears of "Michigan Bronze Wheat" harvested near Stockholm, 1890–93. 1890 was a "yellow rust year"; 1891 no yellow rust; 1892 again "yellow rust year"; and 1893 almost no yellow rust. The seed used was from the crop of the previous year. (Orig.)

For control measures, see below, p. 173.

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Crown Rust of Oats, etc.

Puccinia coronifera Kleb.

This rust attacks oats and a number of grasses. In Sweden the uredo-stage appears on oats rather late in summer, about the end of July or the beginning of August, in the form of round or elongated pustules of an orange-yellow colour on the blades and sheaths of the leaves. The spores are spherical and germinate readily. One or two weeks later the black teleuto-sori, which are covered by the epidermis, appear round the uredo-pustules. The teleutospores bear a circle or crown of digitate projections on their tips—hence the name of the fungus. These teleutospores are true winter spores which after hibernation germinate in the following spring.

In northern countries (Northern and Mid-Sweden) this rust is not important, but in Mid-Europe it is found frequently and appears to be the chief form of rust met with on oats.

The following specialized forms of this rust have been distinguished in Europe: (1) f. sp. Avenæ on *Avena sativa* and *A. brevis;* (2) f. sp. Alopecuri on *Alopecurus pratensis* and *A. arundinaceus*, now and then transmissible to *Avena sativa;* (3) f. sp. Festucæ on *Festuca elatior*, *F. gigantea*, *F. arundinacea* (in Switzerland), *F. varia*, and *F. alpina;* (4) f. sp. Lolii on *Lolium perenne*, *L. temulentum*, *L. rigidum*, *L. italicum*, and *L. remotum* var. *aristatum*, in certain cases transmissible to *Festuca elatior;* (5) f. sp. Glyceriæ on *Glyceria aquatica;* (6) f. sp. Agropyri on *Agropyrum repens;* (7) f. sp. Epigæi on *Calamagrostis Epigeios*, in rare cases transmissible to *Avena sativa;* (8) f. sp. Holci on *Holcus lanatus;* and (9) f. sp. Bromi on *Bromus erectus*, *Br. er.* var. *canadensis*, *Br. inermis*, *Br. sterilis*, *Br. tectorum*, *Br. secalinus*, and *Br. commutatus*.

In Russia (Voronesh Government) an epidemic attack of *Puccinia coronifera* was observed in the autumn of 1924 on winter rye, the crop being very poorly developed. The teleuto-stage was much in evidence, occurring on 85 per cent. of the diseased leaves. Samples consisting of ten plants each were examined on January 8 and March 3, 1925. On the former day seven plants and on the latter day only two plants showed yellow pustules. The spores failed to germinate (Roussakov, 1925).

Studies were made in Wales (Aberystwyth) in 1923–25 on the inheritance of resistance and susceptibility to the crown rust in a number of different varieties of oats. In a cross between resistant selections of *Avena sterilis* and the highly susceptible Scotch potato variety (*Avena sativa*) the F² generation, of 1,041 plants, showed 258 as susceptible and 777 as resistant. Of the latter, 660 showed characteristic flecking, without any pustules, and 117 developed weak uredo-sori. Segregation appears to occur in the F² generation in the ratio of three resistant to one susceptible. In continuation of these experiments some evidence was obtained as to the existence of a certain degree of linkage between various undesirable grain characters and resistance to crown rust. Such an association would greatly complicate the work of selection and might even preclude the possibility of breeding rust-resistant types equal in quality to the best varieties of *Avena sativa*.

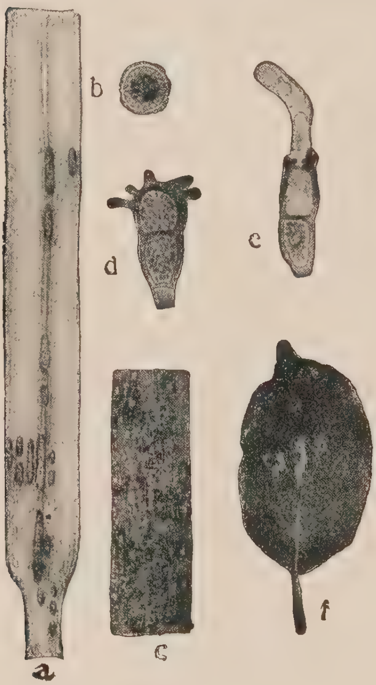

FIG. 116.—Crown Rust of Oats.

a, oat leaf with uredo-pustules; *b*, summer spore; *c*, oat leaf with puccinia-patches; *d* and *e*, winter spores, one in process of germination; *f*, leaf of buckthorn with æcidia. (Orig.)

If the teleutospores after over-wintering germinate on a young leaf of *Rhamnus cathartica* a form of cluster cup (*Æcidium Catharticæ*) arises thereon within 8–10 days. The æcidiospores germinate readily and produce uredo-pustules on those cereals and grasses to which the form in question is adapted.

It has been pointed out, from France, that under natural conditions there is an apparent lack of relationship between the intensity of this rust and the occurrence of its æcidia, as *Rhamnus cathartica* is frequently absent near oat-fields badly infected by the crown rust (Ducomet, 1926). The same experience has been met with in many other places, for instance, in Sweden.

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Puccinia coronata (Corda) Kleb. is distinguished morphologically from the above-mentioned crown rust by the almost total absence of the annular disposition of the teleuto-sori about the uredo-sori. This species develops its æcidial stage (*Æcidium Frangulæ*) on *Rhamnus Frangula*. The following specialised forms have been distinguished: (1) f. sp. Calamagrostis on *Calamagrostis arundinacea* (? *C. lanceolata*, *C. phragmitoides*, *C. calybœa*), in rare cases transmissible to *Phalaris arundinacea*; (2) f. sp. *Phalaridis* on *Phalaris arundinacea*, in rare cases transmissible to *Calamagrostis arundinacea*; and (3) f. sp. *Agrostis* on *Agrostis vulgaris* and *A. stolonifera*. Whether the forms on *Holcus mollis* and *H. lanatus* (? f. sp. Holci), on *Agropyrum repens* (? f. sp. Agropyri), and on *Melica nutans* (? f. sp. Melicæ) are independent specialized forms cannot yet be decided as a sufficient number of inoculation tests have not been made (Eriksson, 1908).

American Crown Rust of Oats

Puccinia coronata Corda

This rust is morphologically identical with the European crown rust, but differs from it biologically in specialization and in the change of host plant.

The Gramineæ chiefly attacked by this crown rust are: *Avena sativa*, *Calamagrostis canadensis*, *Lolium perenne*, and *Holcus lanatus*. The form on oats is transmissible to 16, the form on *Calamagrostis* to 14, and the form on *Lolium* to 13 other species of grasses, whereas the form on *Holcus* is only transmissible to the same species.

Even between the different varieties and lines of *Avena sativa* itself a physiologic specialization has been detected. At the University Farm, St. Paul (Minnesota), 27 forms of cultivated and wild oats were inoculated with collections of crown rust (*Puccinia coronifera avenæ*) from 15 different localities in the United States and Canada. On the basis of the reaction of these varieties to the different collections of rust, it was possible to distinguish 4 (possibly 5) different biologic forms. These forms produce varying effects on the inoculated plants. The forms also appear to differ in their readiness to produce teleutospores, this process being apparently due to an inherent tendency within the strains.

The American crown rust forms on the oat are especially worth consideration in respect of the heterœcism of the fungus. According to artificial inoculations made during the years 1921--26 in the greenhouse and laboratories of the Iowa Agricultural Experiment Station, a more or less perfect æcidial stage—varying between grade 1 (necrotic area, no sporulation), grade 2 (normal pycnidia, no æcidia), grade 3 (normal pycnidia, few æcidia), and grade 4 (normal pycnidia and æcidia)—was produced in the following cases. From *Avena sativa* on *Rhamnus alnifolia*, *Rh. californica*, *Rh. cal.* var. *tomentella*, *Rh. caroliniana*, *Rh. cathartica*, *Rh. crocea*, *Rh. dahurica*, *Rh. frangula*, *Rh. ilicifolia*, *Rh. lanceolata*, *Rh. pachyphylla*, *Rh. pinetorum*, *Rh. purshiana*, *Rh. rubra*, *Rh. Smithii*, and *Rh. tinctoria* (that is, 16 Rhamnus species) and *Berchemia scandens ;* from *Calamagrostis arundinacea* on 15 Rhamnus species ; from *Festuca elatior* on 11 Rhamnus species ; from *Holcus lanatus* on 10 Rhamnus species ; and from *Calamagrostis purpuracens* on *Lepargyrea argentea*.

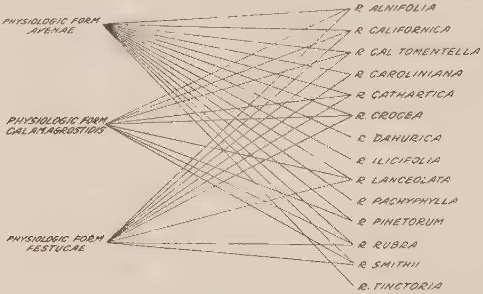
PHYSIOLOGIC FORM AVENAE
PHYSIOLOGIC FORM CALAMAGROSTIDIS
PHYSIOLOGIC FORM FESTUCAE
R ALNIFOLIA
R CALIFORNICA
R CAL TOMENTELLA
R CAROLINIANA
R CATHARTICA
R. CROCEA
R DAHURICA
R ILICIFOLIA
R LANCEOLATA
R PACHYPHYLLA
R PINETORUM
R RUBRA
R SMITHII
R. TINCTORIA

FIG. 117.—Diagrammatic representation of the alternate host of three American physiologic forms of *Puccinia coronata*. (After S. M. Dietz.)

As to the effect of the alternate hosts on the physiologic nature of the fungus, the æcidiospores from *Rhamnus caroliniana*, *Rh. cathartica*, *Rh. lanceolata*, *Rh. pinetorum*, *Rh. Smithii*, and *Rh. rubra*, resulting from inoculation with teleutospores from *Avena sativa*, produced normal infection on *Holcus lanatus*. Æcidiospores from *Rh. lanceolatus*, which had been inoculated with teleutospores from *Calamagrostis canadensis*, produced abundant uredospores on oats, while æcidia on *Rh. lanceolata* produced by teleutospores on oats formed numerous uredospores on *Calamagrostis canadensis*. Æcidia collected on *Rh. alnifolia* in the open produced abundant uredinial infection on oats. Æcidiospores from *Rh. lanceolata*, produced by teleutospores from *Festuca eliator*, caused severe infection of *Festuca interrupta*, *Hordeum murinum*, and *Phalaris stenoptera*.

For control measures, see below, p. 173.

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Dwarf Leaf Rust (or Brown Rust) of Barley

Puccinia simplex (Kcke.) Eriks. & Hen.

This rust is found only on barley and the main outbreak of the disease is relatively late in the summer (August). Very small, scattered, bright yellow uredo-pustules occur on the upper surface of the leaf, and later on the lower surface and on the leaf sheaths brownish black streaks. The winter spores are mainly one-celled. Not infrequently the fungus appears in the ears and on the awns. The disease often causes considerable damage in Denmark and Germany.

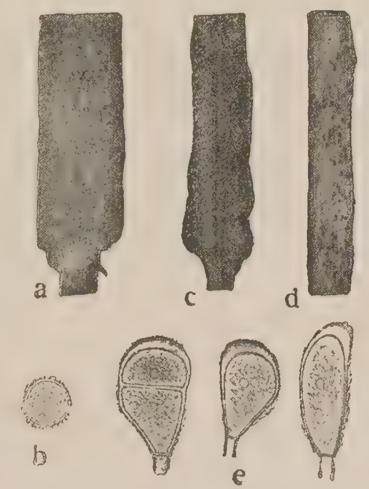

FIG. 118.—Dwarf Rust of Barley. *a*, barley leaf with uredo-pustules; *b*, summer spore; *c*, leaf, and *d*, stem with puccinia-patches; *e*, winter spores. (Orig.)

By artificial inoculation with teleutospores collected in the Crimea in 1909 it was possible to produce the æcidial stage of the fungus on *Ornithogalum umbellatum* (to a considerable extent) and on *O. narbonense*, *Muscari botryoides*, *M. tenuiflorum*, *Scilla sibirica*, and *Allium angulosum*, and with spores from the æcidial stage to obtain uredo- and teleutopustules on *Hordeum vulgare*.

In America (Indiana) in 1922 the æcidial stage on *Ornithogalum umbellatum* was demonstrated under field and greenhouse conditions. In Germany in April, 1927, feebly germinated teleutospores from barley leaves infected with *Puccinia simplex* were laid on the ground in the centre of a group of *Ornithogalum umbellatum* plants, four leaves of which developed yellowish lesions bearing first spermogonia and then æcidia. A young plant of summer barley was successfully inoculated with these æcidiospores, the first uredo-sori appearing in nine days. Finally, in France (Grignon) in 1926 the æcidial stage on *Ornithogalum umbellatum* was produced by artificial inoculation under greenhouse conditions. As, however, these cluster cup forms have only rarely been observed in the open, this stage in the life-cycle of the fungus can hardly play a practical part.

In Australia, glasshouse tests were made in 1926 with 117 varieties of barley, belonging to 6 species, to determine their reaction to an Australian physiologic form collected at the Weiribee Research Station, Victoria. Fifteen varieties of *Hordeum vulgare* proved highly resistant, but none of these was found to be entirely suitable for extended cultivation under New South Wales conditions. In 1923 and the following years a number of crosses were made between susceptible commercial varieties and certain of the above-mentioned varieties. In the F^2 generation segregation occurred in the ratio of three resistant plants to one susceptible one.

For control measures, see below, p. 173.

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Puccinia Maydis Bér., Maize Rust, occurs on *Zea Mays* in the form of elongated, raised, brown patches of uredo-sori on both sides of the leaves; these patches are soon accompanied by elongated, black teleuto-sori in covered streaks. The fungus is probably endemic in America; in Europe it was first observed in Holland in 1837; now it is found in all countries where maize is grown.

At various times the fungus has been transmitted to different species of *Oxalis* on which it formed æcidia. Thus in America in 1904 to *Oxalis cymosa*, in 1905 in Russia to *O. corniculata*, in Austria in 1912 to *O. stricta* and *O. tropæoloides*, and again in America (Iowa) on *Oxalis corniculata* in 1925. In the last-mentioned place, normal infection was produced on *O. corniculata*, *O. europæa*, and *O. tropæoloides* by artificial inoculation with teleutospores from maize, but on *O. valdiviensis* and *O. cernua* only pycnidia.

In 1926, inoculation experiments, carried out in America (Minnesota) with two physiologic forms of the fungus on a large number of selfed lines of maize, showed that one of these lines was susceptible to one rust and resistant to the other, while another line acted in the opposite way. The continuation of the studies in the same place has shown that of 171 selfed lines of maize some were extremely susceptible, others highly resistant, and a further number intermediate. Numerous collections of *Puccinia Maydis* obtained from different places in North America were used for inoculations on eight selfed lines of maize. The results showed that seven distinct physiologic forms of the fungus could be recognized by their parasitic behaviour on these lines. One form differed from the others in its relatively weak pathogenicity, its unusually pale uredospores, and its failure to produce teleutospores (Wellenstek, 1927; Stakman, Christensen, and Brewaker, 1928).

This fungus, which is usually considered relatively innocuous, caused appreciable damage in 1925 on certain lines of maize in Minnesota, especially on new varieties of the host produced by breeding (Stakman and Christensen, 1926).

No specialization has been observed in the case of this rust; varieties of maize differ, however, in their susceptibility to the fungus.

Witches' Broom Rust of Barberry

Puccinia Arrhenatheri (Kleb.) Eriks.

This fungus gives rise to small, round, yellow uredo-sori on the upper surface of the leaves of *Avena elatior*. On the under surface of the leaf teleuto-sori are found in small numbers and the teleutospores germinate in spring. If the germination takes place on young barberry buds they become infected, and in the following year the leaves on the shoots which grow from these buds are rusted; the period of incubation is thus a whole year. Healthy rosettes of leaves may alternate with diseased ones, which is due to infection not having succeeded on all the

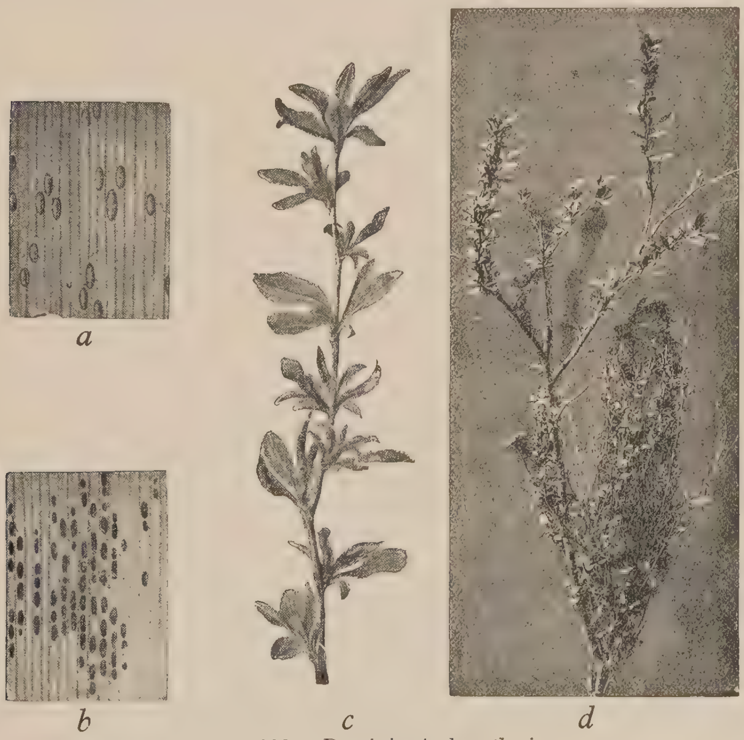

FIG. 119.—*Puccinia Arrhenatheri.*

a, uredo-pustules; *b*, puccinia patches on tall oat grass; *c*, barberry shoot, the buds of diseased leaf-rosettes infected in previous year; *d*, witches' broom on barberry bush. (Orig.)

buds. In the infected buds all the leaves are covered with pycnidia and then with æcidia (*Æcidium graveolens* Shuttl.). The diseased leaves and the twigs on which they are growing remain stunted. The fungus lives in this manner from year to year on the barberry bush and gives rise to the condition known as witches' broom. This species of rust does not infect other grasses.

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Puccinia Poarum Niels. appears on several varieties of *Poa* such as *Poa pratensis*, *P. compressa*, etc. On the upper surface of the leaf it forms small, yellow, scattered uredo-pustules, amongst which occur annular-grouped teleuto-sori covered by the leaf epidermis. The fungus gives rise to an æcidial stage (*Æcidium Tussilaginis*) on the leaves and leaf-stalks of coltsfoot, and it there appears in the form of round, striking, orange-yellow spots. Two biologic forms have been distinguished: (1) f. sp. Poæ pratensis on *Poa pratensis*, and (2) f. sp. Poæ cæsiæ on *P. cæsia* and *P. compressa* (Eriksson, 1923).

Puccinia Poæ-alpinæ Eriks. is found on the upper surface of the leaves of *Poa alpina* in the form of crowded, elongated, uredo-pustules which resemble small pustules of *Uredo graminis*. The teleuto-sori appear on the lower leaf surface and often form large crusts. This form of fungus can also infect—but only feebly—*Poa pratensis* (Eriksson, 1923).

Puccinia Milii Eriks. produces reddish-yellow, afterwards black, pustules on the leaves of *Milium effusum*; the pustules are generally surrounded by large clear areas (Eriksson, 1895, 1923).

Puccinia Anthoxanthi Fuck. appears in the form of small, scattered, yellow pustules on the leaves of meadowsweet (*Anthoxanthum odoratum*).

Puccinia Phragmitis (Schum.) Körn. forms large, elongated lesions (at first brown, later black) on the leaves and panicles of the common reed (*Phragmites communis*). The fungus produces æcidia (*Æcidium rubellum*) on various docks (*Rumex hydrolapathum*, *R. maritimus*, etc.) as well as on rhubarb.

Puccinia Magnusiana Körn. causes very small pustules on the leaves of the reed, but gives rise to æcidia on *Ranunculus repens* and *R. bulbosus*.

Control Measures:

(*a*) *Against Cereal and Grass Rusts generally.*

(1) Avoid, as far as possible, damp, shady, and badly drained fields.

(2) Cultivate the soil by means which will encourage rapid growth and uniform ripening of the crop. (3) Dung should not be applied immediately before spring sowing as it delays maturity. Artificial manures, especially phosphates, should be used as they induce early ripening. (4) The seed should be drilled. (5) Spring corn should be sown early in a well-prepared and suitable soil. (6) Grasses such as the following should be removed from the vicinity of a corn-field; they may carry rust to the young crop: *Triticum repens*, *T. canium*, *Elymus arenarius*, and *Bromus secalinus* (these are undesirable near to rye and barley); *Avena elatior*, *Dactylis glomerata*, *Alopecurus pratensis*, and *Milium effusum* (these are undesirable near to oats).

(*b*) *Against Heterœcious Rusts.*

(7) Remove the host plants that carry the æcidial stage. Such plants are: *Berberis vulgaris*, *Mahonia Aquifolium*, *Rhamnus cathartica*, *Rh. Frangula*, *Anchusa arvensis*, and *A. officinalis*. None of these shrubs should be allowed within a radius of 100 yards of corn- or grass-fields; they should be removed from the sides of roads, railways, and woods as well as from gardens and unused ground; none of the shrubs mentioned should be planted in the danger zone.

(*c*) *Against Yellow Rust.*

(8) A cereal variety which has been known to be attacked by the disease in a so-called "yellow rust year" should not be grown. (9) Even if the variety has only been slightly affected in a year when yellow rust is rare it should not be relied upon, because when the conditions are more favourable to the disease a severe outbreak may result. (10) Reliance should not be placed on the permanent resistance of a variety or stock, as experience has shown that a cultivated sort which has proved resistant for many years may gradually or suddenly change and become badly affected. (11) Should the resistance of a variety begin to break down, another and more resistant one should be chosen without delay, and in choosing such a variety regard should be had to the experience obtained from trials made in the home country. (12) Grains shrivelled through rust injury must in no circumstances be used as seed, for if a "yellow rust year" should occur the crop would certainly be ruined. (13) Resistance to yellow rust should not be the only consideration in choosing a variety of wheat, but other properties such as resistance to cold winters and dry springs, yield, early maturity, etc., should influence the choice.

Experiments have been made in several countries to ascertain whether manuring with potassium salts has value as a protective measure against yellow rust. In Czechoslovakia a crop of wheat receiving a top-dressing in the spring of 150 kg., 40 per cent. potash salts, and 100 kg. ammonium sulphate per hectare, remained almost entirely free from infection, while a similar plot to which only ammonium sulphate was applied showed heavy infection (Hieke, 1927). Similar observations have been made in other places in Europe (Doerell, 1927 ; Günther, 1927 ; Maas, 1927 ; Finger, 1927).

Asparagus Rust

Puccinia Asparagi DC.

This disease appears regularly in late summer—August to September —on the stems of asparagus (*Asparagus officinalis*) as small cinnamon-brown pustules (uredo-sori), which are often joined together to form streaks along the stem. Within a few days small, hard, black cushions (teleuto-sori) containing teleutospores are found. Sometimes in spring and early summer a preliminary æcidial stage of the fungus is observed on the lower parts of the thick asparagus shoots. The pycnidia are yellow and the æcidia are orange, arranged in elongated groups and with a white, jagged margin.

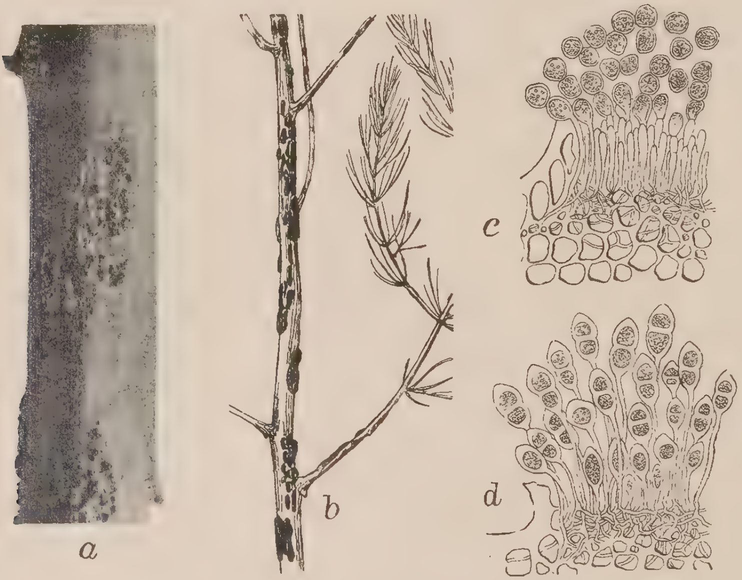

Fig. 120.—Asparagus Rust.
a and *b*, parts of stem attacked by fungus ; *c*, collection of uredospores ; *d*, teleutospores. (*a*, after C. B. Clinton ; *b*, after E. Rostrup ; *c*, after W. C. Sturgis.)

The assimilation of the plant is markedly interfered with by the attack of the fungus, the green colour changes prematurely to yellow, the leaves fall, and in the following year only stunted shoots, some of which fail to reach the surface of soil, are produced; in very severe cases the plants perish.

The fungus was first observed in Europe in 1905 and was then described; it is only during recent years that it has been found to be an injurious parasite, thus in Denmark (1899–1902), Germany (1908), Sweden (1911), Italy (1912), Hungary (1914), etc. The fungus appeared in America at the end of the last century and now causes heavy losses. The first outbreaks occurred in New Jersey, Delaware, Long Island, and New England; the disease then spread southwards and westwards; in 1901 it had reached South Carolina.

In the United States (Indiana) two sorts of asparagus, "Martha Washington" and "Mary Washington," have shown themselves to be resistant to rust (Gregory, 1925). The fungus has been found on *Asparagus capsicus* and *A. maritimus* in America, whilst in Sweden it caused very severe damage to *A. Sprengeri* under glass.

Control Measures:

(1) All parts of the plant showing disease should be cut off at the ground, removed, and burnt. (2) New plants should not be placed too closely together, as a too moist atmosphere must be avoided.

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Puccinia Porri (Sow.) Wint., Leek or Onion Rust, occurs on the leaves, stems, and flower stalks of many species of onion such as *Allium Porrum*, *A. Cepa*, *A. fistulosum*, *A. Schœnoprasum*, etc. At first, bright yellow æcidia, which are often grouped in a ring (at least in the case of certain onions), are produced, and then reddish-yellow uredo-sori and finally leaden-grey, translucent teleuto-sori. These teleuto-sori are powdery, arranged in rows, and remain covered by the epidermis for some time. In the case of certain species of *Allium* such as *A. Scorodoprasum*, the teleutospores are one-celled, and on that account they have also been named *Uromyces ambiguus* (DC.) Fuck. The parts attacked by the fungus turn yellow and fall prematurely. Several biologic forms of the fungus appear to exist (Schneider, 1912; Lind, 1915). The fungus is found principally in Northern and Mid-Europe. In the Mediterranean area, and sometimes also in Mid-Europe, another rust, **Puccinia Allii**, occurs on several varieties of onion ; it is characterized by the absence of an æcidial stage and the teleuto-sori are hard and not powdery (Rytz, 1923).

Puccinia Schröteri Pass. produces teleuto-sori on the leaves of *Narcissus Pseudonarcissus*, *N. poeticus*, and *N. radiiflorus*. The sori are elongated, almost linear, at first covered by the epidermis, which later bursts, brown and powdery.

Puccinia Helianthi Schw. attacks a number—at least sixteen—of different species of the genus *Helianthus*, especially *H. annuus* and *H. tuberosus*, but does not, as far as is known, pass to any other genus of host plants. The fungus can produce all the stages of its development (spermogonium, æcidium, uredo, puccinia) on the same host plant, but it often shows a tendency to omit the æcidial stage. In general, it produces a black dust on the under surface of the leaf. Three, possibly four, biologic forms of the fungus are said to have been distinguished. In certain countries, such as Russia and America, this disease is injurious to the growing of sunflower seed ; from Russia it has spread during the past fifty years to the greater part of Europe (Woronin, 1872 ; Carleton, 1904 ; Bailey, 1923).

Puccinia Cichorii Pass. gives rise to small, cinnamon-brown uredo-pustules on the leaves of chicory (*Cichorium Intybus*) ; occasionally black teleuto-sori appear on the stems.

Puccinia Spergulæ DC. produces brown to black pustules on spurry (*Spergula arvensis*).

Currant Rust

Puccinia Ribis DC.

This fungus appears on the upper surface of the leaves of several species of *Ribes*, such as *Ribes rubrum*, *R. nigrum*, *R. Grossularia*, *R. alpinum*, and *R. petræum*. It forms numerous, scattered sori, each sorus surrounded by a pale, almost white, halo. The red berries become a dirty yellow colour, almost transparent and quite inedible. The fungus develops only one form of spore—two-celled winter spores—which after over-wintering germinate and can infect new leaves and fruit.

In the autumn of 1907 a currant bush heavily infected with rust was placed in the garden at Experimentalfältet (Stockholm) ; in the following year the leaves showed no sign of rust, but in the summer of 1909 a large number of black pustules appeared on the leaves of a shoot which had grown from the root of the bush. From this it may be inferred that the fungus can live inside the stem, either as mycelium or as plasm ; no trace of the fungus could be found in the vicinity.

Biologic forms of the fungus exist ; thus in Sweden in 1896 a form which appeared on *R. rubrum* could only be transmitted to *R. rubrum* and not to *R. nigrum.*

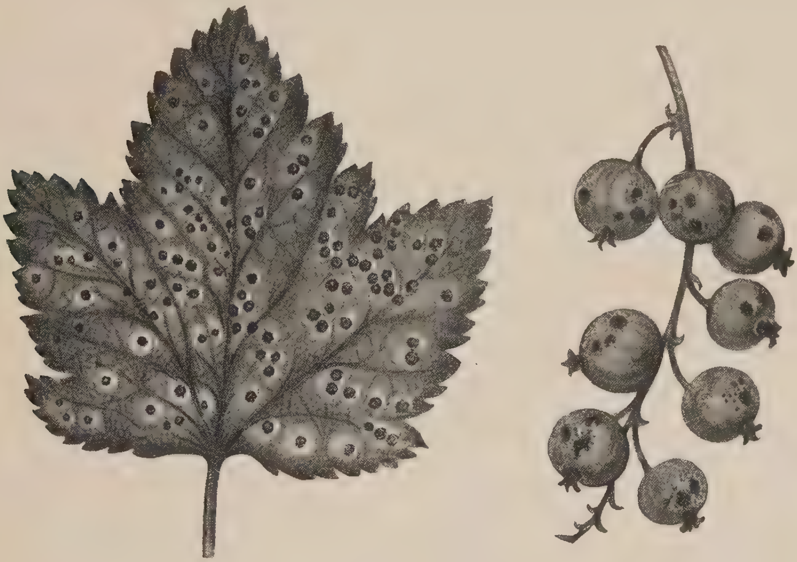

FIG. 121.—Currant Rust. (Orig.)

Since the first appearance of this fungus at the beginning of the last century it has been the cause of much damage, as in Denmark (1890), Germany (1891), Sweden (1896, 1897, 1922), and Norway (1923).

Control Measures :

(1) Collect and burn all infected leaves and berries in autumn. (2) New cuttings or bushes should only be obtained from nurseries that are known to have healthy stocks.

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A number of Puccinia diseases are found on umbelliferous plants ; some of them develop their various spore stages on a single host (autœcious), thus **Puccinia Apii** Desm., Celery Rust, produces, at first, yellowish pycnidia and æcidia on the under surface of the leaves of

Apium graveolens, later dot-like brown to black uredo- and teleuto-sori; the disease is spread through the seed. **P. Cherophylli** Purt., in which the spore-masses are found mainly on the under surface of the leaf. The fungus has a specialized form on *Cherophyllum aureum* and another on *Anthriscus vulgaris* and *A. cerefolium.* **P. Pimpinellæ** (Strauss) Mart. first develops groups of pycnidia and æcidia on the hypertrophied leaf veins of *Pimpinella Saxifraga*, *P. magna*, *P. nigra*, and *P. peregrina.* Later on, brownish-black uredo- and teleuto-sori appear on the under surface of the leaves. The three following species have no æcidial stage: **P. Petroselini** (DC.) Lindr. with a specialized form on *Anethum graveolens*, *Coriandrum sativum*, *Æthusa Cynapium*, *Seseli glaucum*, *S. Pallasii*, *S. coloratum*, and *Libanotis sibirica.* **P. Libanotidis** Lindr. on *Libanotis montana* and *L. sibirica.* **P. Angelicæ** (Schum.) Fuck. on *Angelica silvestris*, *Archangelica officinalis*, *A. atropurpurea*, *A. littoralis*, and *A. decurrens.* The following has no uredo-stage: **P. Falcariæ** (Pers.) Fuck. on *Falcaria Rivini*, with pycnidia. They have a honey-like smell, form æcidia, which are scattered in large numbers over the whole leaf surface, and cause marked deformation of the leaf; finally teleuto-sori arise on both sides of the leaf of *Falcaria Rivini.*

Some other forms of Puccinia that occur on umbelliferous plants have the spore stages on two distinct hosts (heterœcious), thus **Puccinia Cari-Bistortæ** Kleb. with æcidia on *Carum Carvi*, uredo- and puccinia-masses on *Polygonum Bistorta*, and **P. Mei-mamillata** Sem. with æcidia on *Meum mutellina*, uredo- and puccinia-masses on *Polygonum viviparum* and *P. Bistorta* (Semideni, 1904).

Sedge Rust

Puccinia Caricis (Schum.) Reb.

Æcidium Grossulariæ Gmel. & *Æ. Urticæ* Schum.

This fungus appears on the leaves of a number of species of *Carex.* The lesions are usually found on both surfaces of the leaf, at first, small, light brown uredo-pustules and afterwards somewhat elongated, scattered, dark brown, hard teleuto-pustules. The fungus is heterœcious, it develops pycnidia and æcidia on species of *Ribes.* Species A: **Puccinia Caricis-Ribis**, or on species of *Urtica.* Species B: **Puccinia Caricis-Urticæ,** or on species of *Ribes* as well as on species of *Urtica.* Species C: **Puccinia Caricis diffusa.** On species of *Ribes* the æcidia appear on the leaves and on the berries as purple-red or orange spots, those on the species of *Urtica* as irregular swellings on the leaves, leaf-stalks, and stems.

Under the forms belonging to the Species A, three sub-species can be distinguished, subsp. a: **P. Caricis-Ribis diffusa** with æcidia on *Ribes Grossularia* and *R. nigrum* (more rarely on *R. alpinum, R. aureum, R. sanguineum*, and *R. rubrum*) and the succeeding stages on *Carex Pseudocyperus*; subsp. b: **P. Caricis-Grossulariæ** on *Ribes Grossularia* (more rarely on *R. rubrum, R. alpinum, R. aureum, R. sanguineum, R. nigrum*, and *Urtica dioica*) and the succeeding stages on six species of *Carex* (*Carex acuta, C. Buxbaumii, C. Goodenoughii, C. saxatilis*, and *C. stricta*); and subsp. c: **P. Caricis-Ribis-nigri** with æcidia on *Ribes nigrum* (more rarely on *R. alpinum, R. aureum, R. sanguineum, R. rubrum*, and *R. Grossularia*); and of the succeeding stages three specialized forms, (1) f. sp. Acutæ on *Carex acuta* and *C. stricta*; (2) f. sp. Paniculatæ on *Carex paniculata* and *C. paradoxa*; and (3) f. sp. Ripariæ on *Carex riparia* and *C. acutiformis*.

The forms belonging to Species B have been observed on eighteen different species of *Carex*, those belonging to Species C on eight different species of *Carex* (*Carex acuta, C. Goodenoughii, C. ampullacea, C. cæspitosa, C. maritima, C. rigida, C. saxatilis*, and *C. vulgaris*).

The change of host which is seen in certain forms of this fungus appears to depend not only upon the species of *Carex* which acts as host, but also upon the special local conditions at the place of growth. The forms of fungus collected from one and the same species of *Carex* not infrequently show differences in their change of host. In cases where the fungus develops æcidia on *Ribes* as well as on *Urtica* the intensity of the attack differs from that on the single host. Sometimes the fungus infects both groups of host plants equally strongly, but sometimes there is a preference for one group.

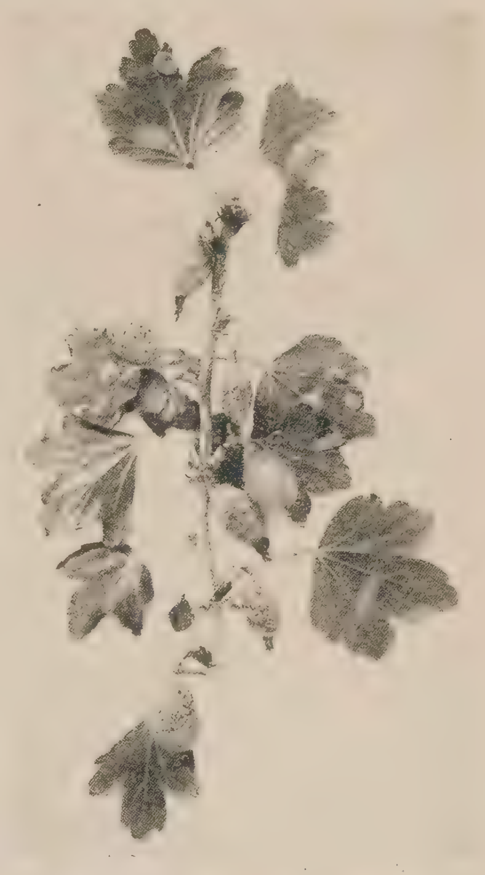

Fig. 122.—Sedge Rust. Æcidial stage on *Ribes Grossularia*. (Orig.)

The heterophagus change of host in the forms of group *C.* appears at first remarkable and unbelievable. The inclination is to attribute the phenomenon to a mixed inoculum (Klebahn, 1893–1922), to errors of experiment or something of that kind. The description given here is, however, based upon very careful inoculation experiments which extended over a period of eight years (1898–1905), and with spore material from varieties of *Carex* obtained from twenty-eight different places in Sweden and Norway. Analogous cases of heterophagus host changes have also been shown to occur with many other species of *Uredineæ.*

Thus **Uromyces lineolatus** Diet., also called *U. Scirpi* (Cast.) Burr., which is present in Europe on *Scirpus maritimus* and in America on *Scirpus americanus, Sc. campestris*, etc., can be transmitted not only to umbelliferous plants such as *Sium latifolium, Berula angustifolia, Pastinaca sativa, Œnanthe crocata, Daucus carota*, etc., but also to *Glaux maritima* and *Hippuris vulgaris* (Dietel, 1895; Plowright, 1890, 1893; Rostrup, 1902; Bubák, 1902; Arthur, 1907–09). The rust **Puccinia subnitens** Diet., which is found on *Distichlis spicata* in America, produced æcidia on 84 species of hosts which belonged to 52 genera and 19 families (Arthur, 1905; Bethel, 1917, 1919). The rust **Puccinia Isiacæ** (Thum.), Wint, which is found on *Phragmites communis* in the Crimea, could be transmitted to 19 species of hosts which belonged to 17 genera and 9 families (Tranzchel, 1906, 1907).

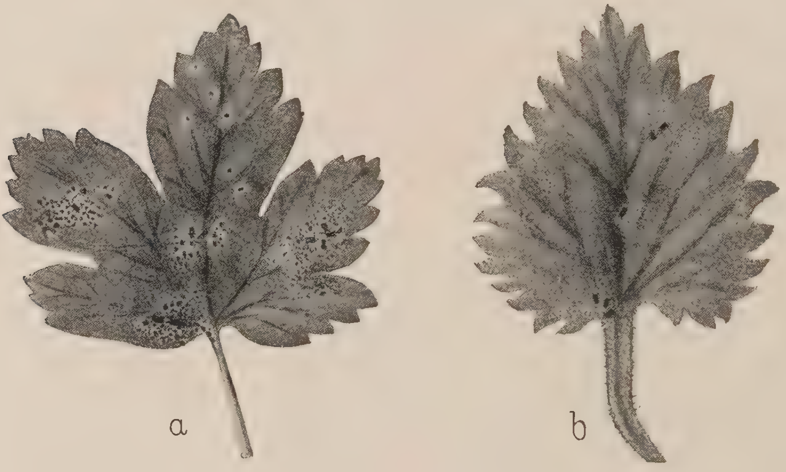

FIG. 123.—Sedge Rust.
Æcidial stage: *a*, on *Ribes Grossularia*; *b*, on *Urtica dioica*. (Orig.)

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Hollyhock Rust

Puccinia Malvacearum Mont.

This fungus appears on the leaves and stems, sometimes also on the sepals and fruits of a number of species of *Malvaceæ*. The hollyhock (*Althæa rosea*) is the most important host plant, then comes *Malva silvestris*. The fungus also attacks many species of hollyhock and mallow and, to a limited extent, individual species of the genera *Malope*, *Sida*, *Lavatera*, *Anoda*, etc. The spread of the fungus from place to place is mainly through the seeds or seedlings from an infected stock.

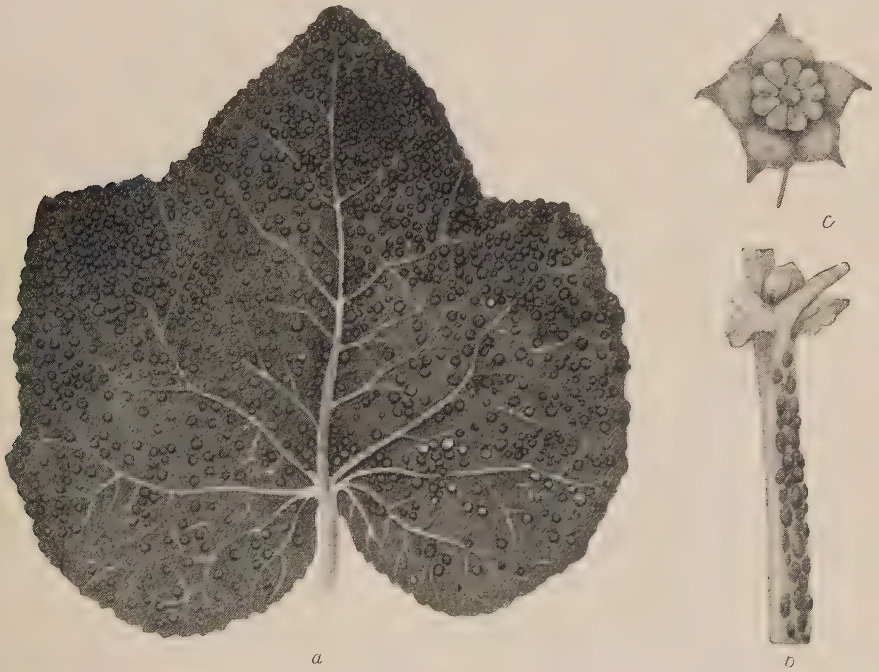

FIG. 124.—Hollyhock Rust on *Althæa rosea*.
a, on under surface of leaf ; *b*, on stem ; *c*, on calyx and fruits. (Orig.)

Hollyhock seedlings grown from seed sown in June remain quite free from rust for the first 2–3 months, whether the seed was infected or not. Seedlings from a healthy stock remain free of disease throughout the year provided they are not infected by a neighbouring diseased stock. If, however, the seedlings are from an infected stock the disease makes its appearance at the end of August or the beginning of September in the form of numerous, raised, chocolate-brown pustules thickly scattered over the whole of the under surface of the leaf. The older leaves are the first to show the disease, the others follow, infection spreading upwards. In a very severe attack all the leaves are shrunken, withered, and pendant; this may be termed the primary autumn outbreak.

The spores of this outbreak are characterized by an "unfixed" germination which may take place in two ways. If the spores are on the surface of a drop of water or in a moist chamber they send out short, broad, curved promycelia which abstrict sporidia: this is termed the "short" germination. On the other hand, if the spores are under water they send out long, narrow, straight threads, the end sections of which are formed into conidia; this is termed the "long" germination.

The sporidia which result from the "short" germination infect the leaf by a small germ-tube pushed through a tiny hole in the outer wall of the epidermal cell into the cell. This germ-tube grows further into the neighbouring palisade cells and intercellular spaces. Such infections, if positive, are followed in 8–15 days by a new crop of pustules.

The conidia which result from the "long" germination apparently infuse their contents as plasm into the epidermal cells. This plasm at first lies as a slimy layer with pointed projections on the outer wall of the epidermal cell: it soon passes, however, to the inner wall and at the same time the cell nucleus, which had lain in a shrunken form on the inner cell wall, enlarges hypertrophically. From the epidermis, the plasm, probably now symbiotically fused with the cell plasm, passes on into the interior of the leaf. After such an infection no trace of new pustules is seen in the following weeks. Leaves upon which the inoculum has been placed remain unchanged and green but are often covered with a slimy mass (probably coalesced plasm exuded by the conidia), or if the inoculum has been used freely there are dead, white areas or holes

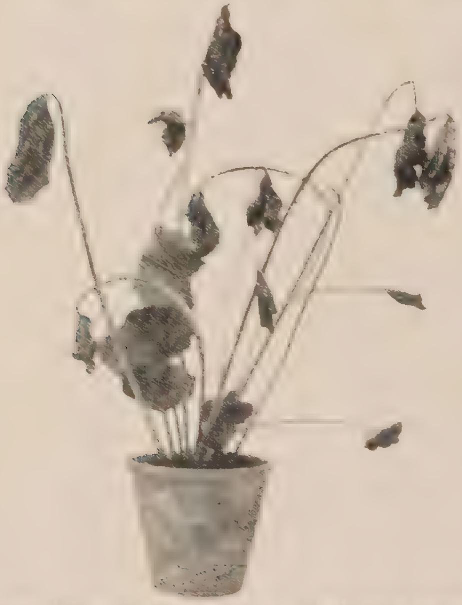

FIG. 125.—Hollyhock Rust. Hollyhock plant raised from seed in June, 1919, attacked by rust at end September, by middle October very severely rusted (from near Stockholm). (Orig.)

The difference in the result of infection by the two forms of spores was strikingly seen in an experiment made in the summer of 1911. The right and left halves of the same leaf were inoculated, one with each spore form; the half on which the sporidia were placed produced brown pustules at the end of a week, on the half inoculated with conidia no pustules were formed.

It is to be assumed, although the further passage of the plasm from the epidermal cell to the interior of the leaf has not yet been traced step by step, that the plasm passes onwards as *Mycoplasm* and finally produces a mycelium because in the tissues lying between erupting sori on the leaves of a diseased hollyhock plant it has been observed, after a time, that there is an extrusion of the fungal substance (separated from the *Mycoplasm*) into the intercellular spaces, in the form of small hyphæ or vesicles.

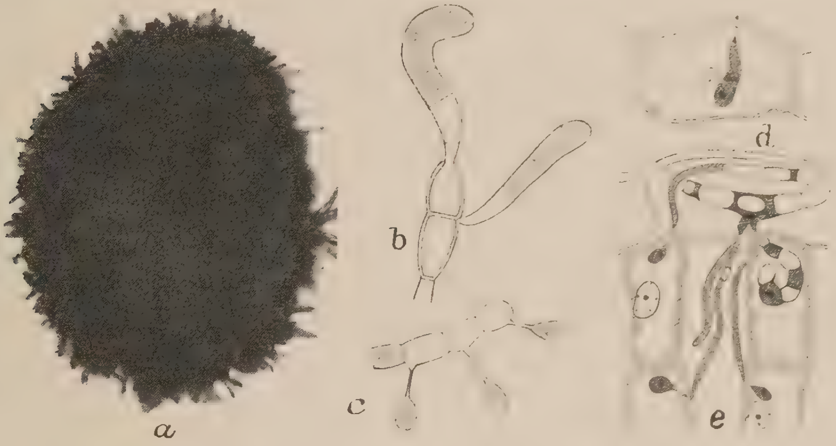

FIG. 126.—Hollyhock Rust, "Short" Germinating Spores.

a, collection of spores; *b* and *c*, single spores abstricting sporidia; *d*, germ-tube entering epidermis, after 24 hours; *e*, dissemination of mycelium after 3 days. (Orig.)

In the second year, when the plant begins to flower, about the end of July, a new outbreak of disease occurs with "unfixed" germination of the spores, and this continues without interruption up to the end of the growing season.

The disease reappears in certain years on over-wintered infected stocks, about the middle or end of May. This summer outbreak usually follows a short interval in the early spring during which there is an absence of the disease. The spores of the summer form are characterized by a "fixed" manner of germination. In moist air or lying under water they germinate only in one way that is, with long hyphæ from the tips of which conidia are abstricted. Generally the summer stage is followed by an autumn stage which begins at the end of July.

In exceptional cases the autumn stage does not occur; thus at Malmö (Sweden) in 1920 a plant of *Malva silvestris* suffered so badly (from the middle of June to the end of the growing period) through the summer stage that all the leaves died and fell.

The over-wintering of the fungus on hollyhock plants infected in autumn is (at least near Stockholm) not by means of spores or mycelium which may have persisted in the remains of leaves or in the hibernating buds, but by a latent plasm (contained in the bud) which has fused symbiotically with the protoplasm of the host cell, the so-called *Mycoplasm*.

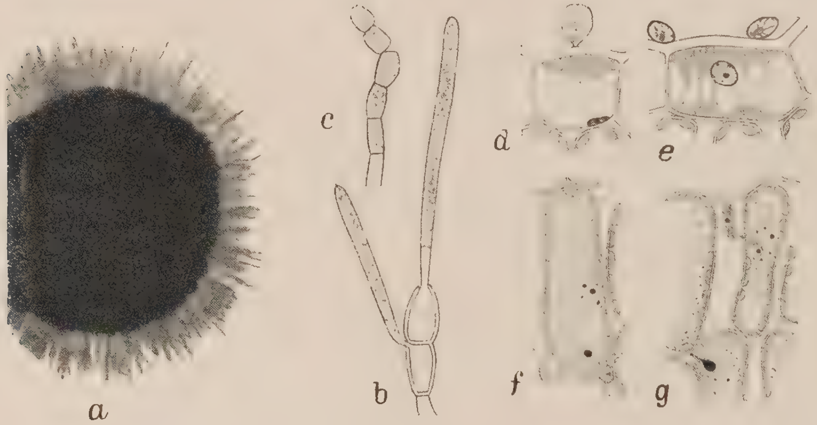

Fig. 127.—Hollyhock Rust, "Long" Germinating Spores.

a, collection of spores; *b* and *c*, single spore abstricting conidia; *d* and *e*, plasm entering epidermal cell, nucleus hypertrophied, after 10 hours; *f* and *g*, fungus material separated from mycoplasm passing into the intercellular spaces.

An exception to this rule was seen in 1913–14 in the case of a stock of hollyhocks near Stockholm. The seedlings, which had been raised from seed in the summer of 1913, were severely attacked by rust in the autumn. The plants were kept in frames covered with lights over the winter. Owing to a spell of abnormally warm weather in April, 1914, the plants grew very quickly and strongly. The first and second leaves, apparently the last of the autumn leaves, were seen to be rusted when the frames were uncovered; the spores were viable from April 18 to July 6.

In 1913, two stocks of hollyhocks of different origin were tested side by side in the vicinity of Stockholm. The plots adjoined and the plants were raised from seed in June, 1912. The seedlings of one stock were severely affected by rust from September 1 to October 30, the other stock remained unaffected during this time. The seedlings were wintered out of doors covered with leaves and boards; on May 2, 1913, thirty-two plants of the diseased stock were planted out in cylindrical zinc vessels and on the same day, thirty-two plants of the healthy stock were planted out in cement boxes; the distance between the two lots was about five yards. The leaves of all the plants were carefully examined ten times between June 3 and August 23 and the amount of rust recorded, the following marking being adopted: Grade 1=1–10 pustules per leaf, Grade 2=10–25, Grade 3=25–100, and Grade 4= more than 100.

Table I given below shows the results, which clearly indicate: (1) that there are two periods in the life of the fungus; (2) that in the first period—from the beginning of May to the end of July—the rust attack is less than in the second period—end of July to the beginning of the cold weather; (3) that the plants from the healthy stock remained free from rust up to the end of July but then suddenly, within two or three days (near Stockholm it was July 28–30), an attack developed on all except the youngest leaves, and the under-leaf surface was thickly covered with chocolate-brown pustules (Grade 4).

Hollyhock Rust near Stockholm in the Summer of 1913.

TABLE I

A HEALTHY STOCK GROWING NEAR TO A DISEASED ONE.

Day.	Diseased stock. 2 cylinders—8 plants.						Healthy stock. 2 boxes—8 plants					
	Number of leaves.											
	Degree of rust.					Dead.	Degree of rust.					Dead.
	0	1	2	3	4		0	1	2	3	4	
June 3 . .	61	1	1	—	—	—	All leaves free.					
June 9 . .	45	15	2	1	—	—	,, ,,					
June 18 .	30	8	14	8	1	2	,, ,,					
June 27 .	21	6	8	21	2	5	,, ,,					
July 4 . .	21	5	3	21	6	7	,, ,,					
July 25 .	12	16	8	24	12	33	,, ,,					
July 29 . .	13	18	13	17	54	36	—	—	—	—	—	—
July 30 . .	—	—	—	—	—	—	28	15	13	18	61	—
August 8 .	—	—	—	—	—	—	30	15	13	15	61	—
August 23 .	35	22	27	10	0	74	13	11	14	25	69	36

It can hardly be doubted that the sudden and violent outbreak of disease on the leaves of the healthy stock was due to external infection from the neighbouring diseased stock, either through the large number of conidia spread by the diseased plants in the period from the beginning of June to the end of July, and which only after some time showed any effect externally, or through the sporidia which developed at the end of July and which, in the course of a week, produced new pustules.

A further experiment made in the summer of 1913 yielded striking and instructive results. In this case the soil was watered with a solution of copper sulphate (strengths of 1–5 per cent. were used). Infected hollyhock plants were placed in eight zinc cylinders let into the ground, four plants to each cylinder; two of the cylinders were used as controls and watered with water only. The remaining six cylinders were watered with copper sulphate solution as follows: 2 cylinders on June 4 with 1 per cent. and on June 10 with 2 per cent. solution, 2 other cylinders from June 14 to August 14 with 3 per cent., and the 2 remaining cylinders on June 18 with 4 per cent. and on June 25 with 5 per cent. The difference in susceptibility to rust of the plants watered with 1–3 per cent. fungicide and those with water only is clearly shown in Table II below.

TABLE II

Reduction in the Amount of Rust through Watering the Soil with Copper Sulphate Solution.

Day.	Copper sulphate solution 1–3 per cent.						Water.					
	Number of leaves.											
	Degree of rust.					Dead.	Degree of rust.					Dead.
	0	1	2	3	4		0	1	2	3	4	
June 3 . .	59	3	0	0	0	0	61	1	1	0	0	0
June 9 . .	48	13	1	0	0	0	45	15	2	1	0	0
June 18 .	47	10	2	0	0	3	30	8	14	8	1	2
June 27 .	45	12	1	0	0	7	21	6	8	21	2	5
July 4 . .	54	4	0	0	0	7	21	5	3	21	6	7
July 25 . .	53	26	0	0	0	21	12	16	8	24	12	33
July 29 . .	33	29	18	19	13	25	13	18	13	17	54	36
August 23 .	60	16	19	7	0	43	35	22	27	10	0	74

The results recorded in Table II have a double interest; on the one hand it is shown that the fungus in the summer, vegetative stage (probably as *Mycoplasm*) in June and July is very sensitive to the action of the fungicide, which suggests the possibility of using an internal therapeutic method for the control of the disease. On the other hand, it is shown that from the beginning of August, when the mycelium stage of the fungus begins, the fungicide is without effect, that is to say, the fungus is not sensitive to any method of internal therapeutic control. Further, it may be pointed out that even a 5 per cent. copper sulphate solution does not affect the growth of the plant nor its green colour. The action of the fungicide was clearly shown in the above experiments, in that black, dead rings were formed round the pustules on the leaves of the plants present watered with the copper sulphate solution, the fungicide taken in through the roots having acted upon the spore-forming hymenial layer which surrounds the pustules.

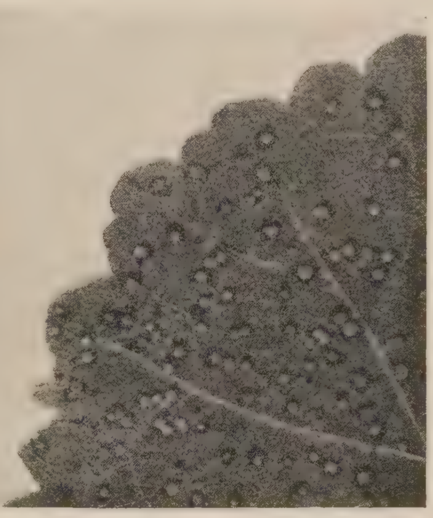

Fig. 128.—Hollyhock Rust. Leaf of hollyhock from one of plants watered with 1·5 per cent. solution of copper sulphate; the pustules surrounded by large, black rings. (Orig.)

It is noteworthy that in the experiments carried out near Stockholm during a period of eight years (1912–26) with 16 different stocks of hollyhocks the conditions, as regards the rust, varied widely. There were found: (*a*) 7 stocks (1912–16) which were diseased from the commencement to the end; (*b*) 2 stocks (1913–14) which were originally healthy but in the spring and summer of the second year of growth were infected by neighbouring diseased plants; (*c*) 4 stocks (1917–20) which behaved throughout as healthy; (*d*) 2 stocks (1918 and 1920) which at first were diseased but finally showed themselves to be healthy. Further, it must be noted that the summer stage of the disease failed to appear, either entirely or partially, in the years 1915–20.

The above account of the nature and life-history of the hollyhock rust is based on investigations and experiments carried out over a period of twenty years (1900–20) with numerous stocks of hollyhocks, and it applies primarily to Swedish conditions of growth and climate. To what extent the conditions in Mid- and Southern Europe may introduce variations must be left to investigators in these countries to decide. As, however, the conditions in the same place may vary so much from year to year—as has been pointed out—it is clear that the results of one or two years' experiments cannot have any general application.

This fungus was first observed on *Althœa officinalis* in 1852 in Chili, and on *Althœa rosea* in Melbourne (Australia) in 1857. Spain (1869) was the first European country in which it was found, and in the following decade it spread to other countries such as Belgium, Holland, Italy, and Denmark (1871), France (1872, 1873), England and Germany (1873), Ireland and Switzerland (1875), Austria-Hungary (1876), Greece (1877), finally reaching Sweden (1882) and Finland (1890). The fungus appeared relatively late in North America (1888). In the last decade of last century and in the first decade of the present one, the disease was general and virulent, especially in all European countries, and it was rare to find a bed of healthy hollyhocks; latterly the disease seems to have become reduced both in distribution and virulence. The disease can only be avoided by using seed which it is known has come from healthy plants.

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Chrysanthemum Rust

Puccinia Chrysanthemi Roze.

This fungus chiefly attacks cultivated forms of *Chrysanthemum indicum* grown under glass. Chocolate-brown uredo-sori appear either separately or in circular groups on the under surface of the leaves, more rarely on the upper surface also. The fungus is found on brown spots which arise on the leaf and gradually enlarge until the leaf is finally killed.

The one-celled uredospores are spherical or pear-shaped with a light brown, echinulate wall; sometimes, also, two-celled uredospores (twin uredospores) are found in large, well-developed sori. These twin uredospores agree exactly in colour and nature of the spore wall with the one-celled spores and they have probably arisen by fusion. It is to be noted that the two-celled spores always germinate vegetatively without the formation of sporidia; their germ-tubes are partially septate and may be of a length of 200 μ or more. The incubation period, after inoculation with uredospores, is unusually long, at least 3–4 weeks. Whether the two-celled spores play a different part in the economy of the fungus than do the one-celled spores is not known.

The true teleutospores, which are chestnut-brown and covered with small warts, have occasionally been found in Europe in a stunted form associated with vigorous uredo-sori, but in Japan, where the fungus is endemic, they develop strongly.

The young shoots which arise in autumn from the roots of the chrysanthemum become infected from the rusted leaves, and, in consequence, if these shoots are used as sets and over-wintered under glass they carry on the disease. The fungus appears to be able to hibernate in the uredo-stage and so the possibility of young sets becoming infected in the spring through material which has remained in the open must be considered.

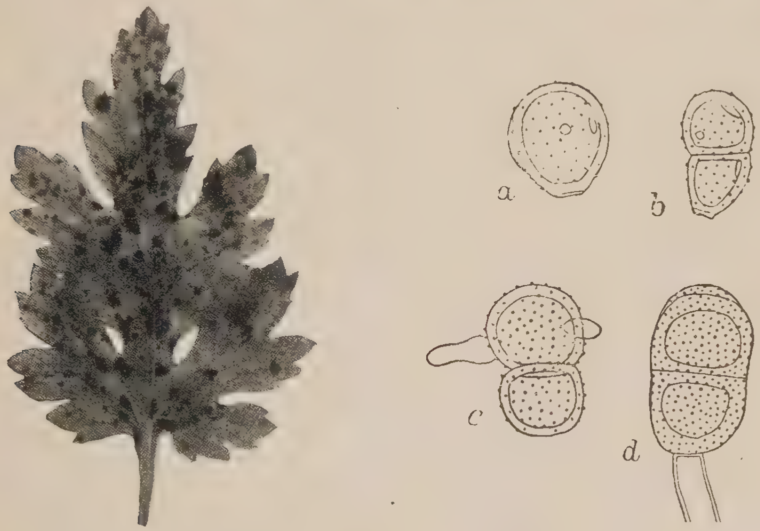

Fig. 129.—Chrysanthemum Rust.

a–c, spores : three uredospores, one unicellular, one bicellular ungerminated, one bicellular germinating ; *d*, an ungerminated teleutospore. (*a–d*, after E. Jacky.)

The fungus has been very prevalent in Japan for many years past ; in Europe it was first observed in England in 1895. In 1897 or 1898 it appeared in Mid- and Northern Europe and was found in France, Italy, and Denmark (1897), Germany (1898), Holland (1903), Sweden (1905), and South Africa (1921), etc.

Control Measures :

(1) Every diseased leaf should be removed and burnt. (2) Badly infected plants should be destroyed. (3) Diseased plants should be

isolated. (4) Shoots from the roots of infected plants should not be used as sets in the next year. (5) Care should be taken not to obtain chrysanthemum plants from nurseries where the disease exists.

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Puccinia Buxi DC. produces isolated, round, cushion-like, domed teleuto-sori on the leaves of *Buxus semperflorens*. On the upper surface of the leaf the sori are bright green, on the lower surface white. In late autumn, or in the course of the winter, spores are formed which mature in the following spring and infect the tender bright green leaves of the previous year; in these leaves the mycelium slowly develops. The period of incubation appears to be a long one, 2–3 months. During the summer and autumn the infected patches on the leaves become increasingly thickened and enlarged (E. Fischer, 1901).

Puccinia Liliacearum Duby. In the spring the fungus gives rise to yellow, conical, raised pycnidia on the leaves of *Ornithogalum umbellatum*, *O. pyrenacium*, *O. nutans*, *O. narbonensis*, etc., and soon afterwards teleuto-sori, which are dark brown on both sides. The spores hibernate in the surface soil and infect the young leaves in the following spring (E. Fischer, 1905). Forms nearly related to this fungus are found on species of *Narcissus*, *Muscari*, and *Hyacinthus*. **P. Galanthi** Ung. produces chocolate-brown teleuto-sori which arise on both sides of the leaves of *Galanthus nivalis*. The spore wall usually has raised, longitudinal thickenings. The fungus has been observed in Austria (Bubák, 1897). **P. Iridis** (DC.) Wallr. develops on the leaves of a number of species of *Iris*, such as *I. graminifolia*, *I. pumila*, *I. germanica*, *I. nudicaulis*, etc. In certain cases, only uredo-sori, which are able to continue over the winter, are formed, but in other cases teleuto-sori also. The fungus is widely found in European countries. **P. Gentianæ** Strauss. is found on the leaves of numerous species of *Gentiana* such as *Gentiana acaulis*, *G. cruciata*, *G. asclepiadea*, etc. In spring pale- to orange-yellow pycnidia and æcidia are produced, whilst in summer and autumn brownish-yellow uredo-sori and blackish-brown teleuto-sori generally appear. This fungus, which is disseminated throughout Europe, shows no specialisation (Bock, 1908).

Puccinia fusca (Pers.) Wint. forms black, dusty teleuto-sori on the leaves of species of *Anemone* and *Pulsatilla*, e.g. *Anemone nemorosa*, *A. silvestris*, and others, *Pulsatilla vulgaris*, *P. patens*, etc. The rust-sori are mainly on the under surface of the leaf and regularly distributed. Infected leaves are often misshapen and die prematurely. This species, which is also named *Puccinia Pulsatillæ* Kalchbr., has been shown to have several biologic (specialised) forms: (1) f. sp. *genuina* on *Pulsatilla patens*, infects also *Anemone silvestris*; (2) f. sp. *montanæ* on *Anemone montana*, *A. vernalis*, *A. pratensis*; (3) f. sp. *pulsatillarum* on *Pulsatilla vulgaris* and *P. pratensis*; and (4) f. sp. *contortica* on *Pulsatilla alpina* and *P. sulphurea*. The form on *Anemone ranunculoides* is usually regarded as a separate species, **Puccinia singularis** Magn. and the form on *Atragene alpina* as **Puccinia atragenicola** (Bub.) Syd (Bubák, 1901; E. Fischer, 1913).

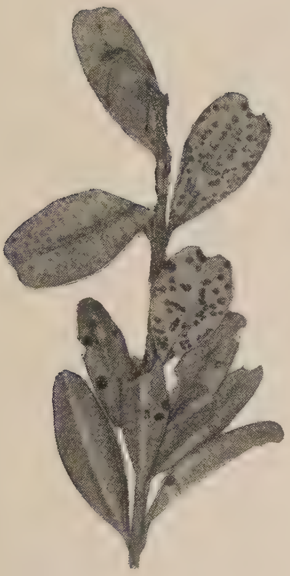

FIG. 130.—*Puccinia buxi.* (Orig.)

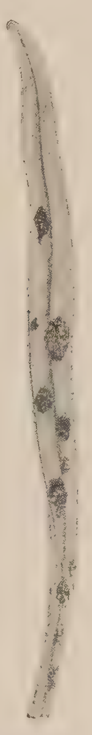

FIG. 131.—*Puccinia galanthi.* (After F. Bubák.)

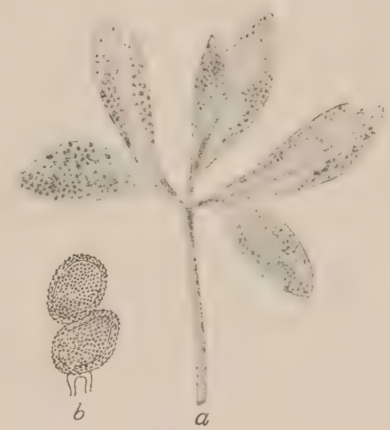

FIG. 132.—*Puccinia fusca.* *a*, diseased leaf; *b*, teleutospore. (After F. Bubák.)

Several species of rust are found on species of *Viola*. **Puccinia Violæ** (Schum.) DC. gives rise in spring to raised spots on the leaves, leaf-stalks, and stems of *Viola odorata*, *V. silvatica*, *V. stagnina*, *V. tricolor*, etc. Pycnidia and æcidia arise from these spots, and later, chiefly on the under surface of the leaf, light brown uredo-sori and brownish-black teleuto-sori are found. **P. depauperans** (Vize) Syd. forms æcidia and teleuto-sori, often both at the same time, on *Viola cornuta*, *V. lutea*, and *V. tricolor*. **P. alpina** Fuck. appears on *Viola biflora* in the form of round or oblong brownish-black teleuto-sori.

Puccinia Centaureæ DC. at first produces orange-red pycnidia and æcidia on both sides of the leaves of numerous species of *Centaurea*, later brown to black uredo- and teleuto-sori appear on the under surface. At least ten different biologic forms of this fungus have been described (Hassler, 1918). **P. Asperulæ-odoratæ** Wurth. first appears as yellow æcidia, afterwards as small, scattered, chocolate-brown uredo- and teleuto-sori, on the under surface of the leaf and on the stems of *Asperula odorata*. Closely-related forms are found on *Asperula cynanchica* and on a number of species of *Galium* (Wurth, 1905). **P. Menthæ** Pers. is found on numerous species of *Mentha* and also on species of the genera *Satureya*, *Thymus*, *Calamintha*, *Clinopodium*, *Origanum*, etc. In spring, pycnidia and æcidia appear on deformed stems and leaf-stalks, sometimes also on the main leaf veins. In summer and autumn brown uredo- and brownish-black teleuto-pustules arise. The disease injures peppermint plants which are to be used for the industrial extraction of peppermint or menthol by checking the development of the oil-producing glands (Ross, 1924). A number of biologic forms have been distinguished in the case of this fungus, four are found on species of *Mentha* and three on species of *Satureya*. The following are regarded as separate species : **Puccinia Glechomatis** DC. on *Glechoma hederacea*, **P. Salviæ** Ung. on *Salvia glutinosa*, **P. Stachydis** DC. on *Stachys annua*, and *St. erecta* and **P. annularis** (Str.) Schl., with (1) f. sp. *Chamedryos* on *Teucrium Chamedrys*, and (2) f. sp. *Scorodoniæ* on *T. Scorodonia*.

Certain æcidial forms observed on *Labiatæ* have been classified under the heterœcious grass rusts, for example : **Puccinia Brunellæ-Moliniæ** Auch. and **P. Thymi-Stipæ** Kleb. (Cruchot, 1926). **P. Antirrhini** Diet. & Holw. produces brown pustules, which contain both uredo- and teleutospores, on the leaves and stems of *Antirrhinum majus*. Inoculations with hibernated, viable teleutospores gave negative results, from which it has been concluded that the fungus is heterœcious. The fungus has wrought much havoc in the United States (Dickson, 1921 ; Mains, 1924).

Puccinia Pruni-spinosæ Pers. forms cinnamon-coloured uredo-sori and dark brown teleuto-sori on the under side of the leaves of some species of *Prunus* and *Amygdalis*. The uredospores are pear-shaped and thickened at the apex. Between these spores are paraphyses, the heads of which are club-shaped. The teleuto-sori, which often cover the whole of the under surface of the leaf, are powdery. The fungus forms pycnidia and æcidia on *Anemone ranunculoides* and *A. coronaria;* this stage was formerly named *Æcidium punctatum* Pers. (Tranzschel, 1905). The pycnidia occur on both sides of the leaf, whereas the æcidia are scattered and only found on the under surface. The infected leaves are stunted and shrivelled. The change of host appears to be only facultative in the case of this fungus as the uredospores from *Prunus* leaves and the mycelium of the *Anemone* fungus are believed to be perennial in the root stock of the host plants and to germinate in the following year; both stages are independent of one another.

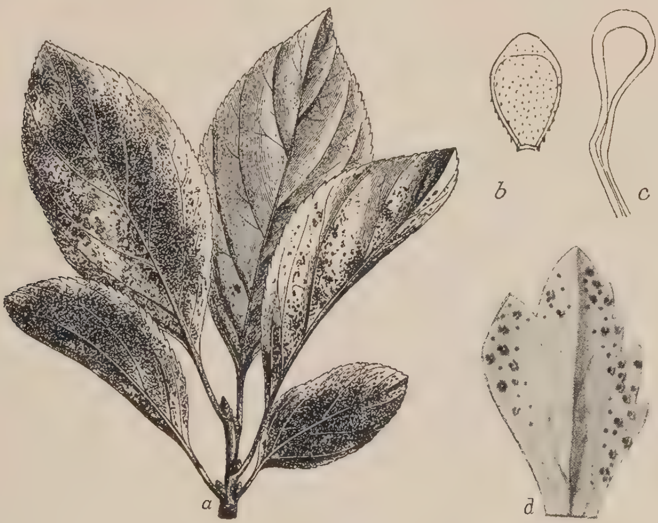

FIG. 133.—*Puccinia Pruni-spinosæ.*

a, diseased leaf of *Prunus domestica ; b,* uredospore ; *c,* paraphysis ; *d,* æcidial stage on *Anemone ranunculoides.* (*a,* after P. Dietel ; *b* and *c,* after E. Fischer ; *d,* after F. Bubák.)

Two forms are distinguished in the *Prunus* fungus : (1) f. sp. *P. typica,* both cells of the teleutospore are of the same size and deep colour; this form arises on *Prunus spinosa, P. insititia,* and *P. armeniaca;* and (2) f. sp. *discolor,* the lower cell of the teleutospore is narrower than the upper one and of a lighter colour; this form is found on *Prunus persica* and *Amygdalis communis,* sometimes also on *Prunus armeniaca,* rarely on *P. domestica, P. spinosa,* and *P. insititia* (Jacky, 1901). Probably the *Anemone* fungus has also different forms, one on *Anemone ranunculoides,* which infects *Prunus spinosa,* and the other on *Anemone coronaria,* which infects *Amygdalis communis, Prunus spinosa,* and *P. divaricata.*

This fungus is known throughout the world and has caused much damage in Australia, where it attacks the fruit, and even the stems, of young trees growing rapidly in the nursery (McAlpine, 1895, 1902, 1906 ; Cunningham, 1922).

For the control of the disease it is recommended to collect and destroy all rusted leaves in autumn and to spray with fungicides when the buds are opening in the spring and again at a later date, so as to provide protection against fresh attacks of the fungus.

This æcidium must not be confused with *Æcidium leucospermum* DC., which is found on some species of *Anemone* and is placed together with *Ochrospora Sorbi* (Qud.) Diet.

Puccinia Saxifragæ Schlecht. produces, usually on the under surface of the leaf, crowded, dark brown, powdery teleuto-sori on the leaves of such Saxifrages as *S. rotundifolia*, *S. granulata*, etc. The spores can germinate after over-wintering or also at once after they arise during the summer. There appears to be specialisation with this fungus as the form on *Saxifraga stellaris* when used for inoculation experiments was only transmitted to this host and not to *S. rotundifolia* and *S. androsacea* (E. Fischer, 1912). **P. Veronicarum** DC. is found on the under surface of the leaf of *Veronica longifolia*, *V. spicata*, *V. urticifolia*, etc., as teleuto-sori which are arranged in groups or sometimes in rings. The sori early become naked, brown, and powdery in the form *fragilipes* or grey, hard, and pulvinate in the form *persistens*. The teleutospores of the first form just mentioned are rather brightly coloured and have a fragile stalk ; they do not germinate at once on ripening. The teleutospores of the other form have a thin, colourless, or yellowish membrane and a substantial stalk ; after ripening they germinate at once on the living plant. The two forms of spores play different parts in the life-history of the fungus. In the case of the form *fragilipes* the spores function as resting spores and enable the fungus to over-winter. In spring these spores germinate and produce fresh sori which, however, contain only spores of the form *persistens*. The latter spores serve as summer spores and take the place of uredospores which in this case are not produced. A little later on, sori, containing spores of the form *fragilipes* (by means of which the fungus over-winters), are formed around the sori containing spores of the form *persistens*, and in this way hibernation is provided for (E. Fischer, 1904).

Uromyces

In the case of the species of rusts belonging to this genus the winter spores are one-celled, usually egg-shaped, and collected in open sori.

Mangel and Beet Rust

Uromyces Betæ (Pers.) Kühn.

The æcidial stage of this fungus can sometimes, although relatively rarely, be observed on young seedlings of sugar beet and mangels as well as on the shoots of plants which are being grown for seed. The lesions are sharply defined and of a bright yellow colour. As a rule the disease does not appear in the field until much later—during August—and it is then in the form of small, brown spots scattered over the whole surface of the leaf. These spots contain the summer spores which can germinate at once and spread the disease over the field. Very soon the light brown spots are replaced by dark brown ones which contain the winter spores of the fungus.

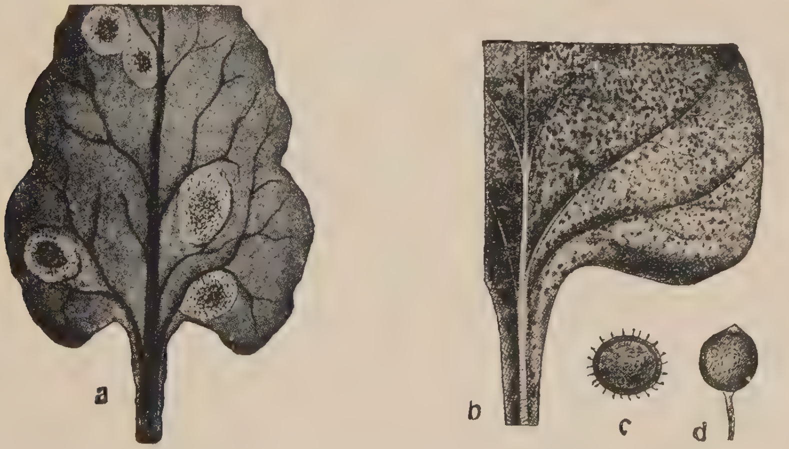

Fig. 134.—Mangel and Beet Rust.

a, leaf of beet with æcidia ; *b*, leaf of beet with uredo- and puccinia-pustules ; *c*, summer spores ; *d*, winter spores. (After O. Kirchner and H. Boltshauser.)

It is very noticeable that neighbouring plants are attacked by the fungus to different extents. In spite of the available possibilities of infection, plants in the vicinity of a badly rusted one may remain healthy, or nearly so, throughout the growing period. This circumstance, as well as the strikingly late appearance of the outbreak in summer, leads to the view that there is here an inherited internal cause of the disease.

On badly infected plants the leaves droop prematurely, the older ones becoming withered and dead. Although the stalks of the younger leaves remain erect the blades are crumpled, drooping, and yellow.

The disease, which was almost unknown in the fifties of the last century, has now spread to all beet-growing countries. It has also been found on garden (red) beet and on wild beet (*Beta maritima*) growing on the west and south coasts of Europe.

Control Measures :

(1) Seed should be obtained from a district in which there is no disease. (2) Fields on which beet is grown for seed should not be near the real beet-fields.

In the United States (Arkansas Valley, Colorado) an æcidial form, which belongs together with the rust *Puccinia subnitens* Diet. on *Distichlis stricta*, was observed on sugar beet in 1912 and 1913 (Pool and Kay, 1914).

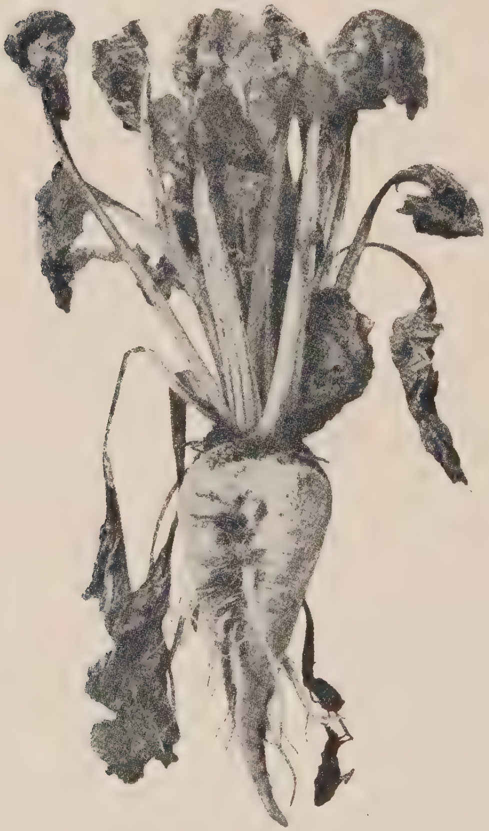

FIG. 135.—Sugar Beet Plant severely attacked by *Uromyces Betæ*. (Orig.)

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Amongst the forms of *Uromyces* which are found on leguminous plants may be mentioned : **Uromyces Fabæ** (Pers.) de By. on horse, or field, beans (*Vicia Faba*). The cluster cup (æcidial) stage gives rise to small rings on the under surface of the leaf ; it is, however, rare. On the other hand, the cinnamon-brown uredo-pustules which appear during the height of summer are present in large numbers. Somewhat later, the hard, brownish-black, elongated teleuto-sori make their appearance. Similar forms of rust appear on peas, tares, lentils, etc. **U. Trifolii-repentis** (Cast.) Liro is found on white clover. The æcidia break out on the leaf-stalk and on the veins, producing small lesions ; they also appear on the leaf between the veins in the form of small rings. The brown to black uredo- and teleuto-sori are produced later, mainly on the under surface of the leaf (Köbel, 1920). **U. Trifolii** (Hedw.) Lév. is very similar to the previous rust but has no æcidial stage; it is found on *Trifolium pratense* and *T. medium*. **U. Anthyllidis** (Grev.) Schröt. produces rust-brown, later black, lesions on the leaves and stems of *Anthyllis Vulneraria*. **U. Onobrychidis** (Desm.) Lév. forms brown, ultimately black, encrusted groups on the leaves and stems of sainfoin (*Onobrychis sativa*). **U. striatus** Schr. is found on *Trifolium arvense*, *T. agrarium*, etc., also on *Medicago sativa*, *M. falcata*, *M. lupulina*, etc., produces æcidia on *Euphorbia Cyparissias*.

Amongst the forms of *Uromyces* which are found on grasses, the following may be mentioned: **Uromyces Dactylidis** Otth. gives rise to very small, crowded yellow, afterwards black, lesions on the leaves and leaf sheaths of cocksfoot (*Dactylis glomerata*). The æcidial stage of the fungus appears on species of the genus *Ranunculus* such as *R. polyanthemos*, *R. bulbosus*, *R. repens*, and *R. acer* (Krieg, 1907, 1910). **U. Poæ** Rab. produces similar lesions on *Poa trivialis*, *P. nemoralis*, and *P. palustris*, and develops an æcidial stage on *Ranunculus repens* and *R. bulbosus* (Semideni, 1910).

Bean Rust

Uromyces appendiculatus (Pers.) Link.

This disease attacks a number of varieties of beans. Almost all the sub-varieties of climbing beans, *Phaseolus vulgaris*, are very susceptible, whilst the dwarf beans, *Ph. vulgaris nanus*, are less susceptible, and the sub-varieties of *Ph. multiflorus* are almost immune. The disease usually appears in the summer when the bean plants have reached a considerable size. The brown uredo- and black teleuto-sori are produced on each side of the leaf, and both of these sori liberate a sooty dust which spreads over the field. Lesions sometimes appear also on the pods.

An æcidial stage only occurs rarely, either early in the year (May) in the form of a few isolated spots, or more generally in summer (August) about the same time as the uredo- and teleuto-stages and in larger numbers. Without doubt, the outbreak of the æcidium in spring cannot be the result of infection by the over-wintered teleutospores of the previous year. How the late outbreak of æcidia, which occurs almost regularly, is to be explained is a difficulty. The period of incubation following an inoculation with teleutospores is limited to 1–2 weeks (de Bary, 1863; Plowright, 1889), whereas the late outbreak of æcidia does not take place before the beginning or middle of August, that is, at least two months after the growing bean seedlings were susceptible to external infection. It must be assumed, therefore, that this outbreak of disease comes from some infective material (a mycelium or plasm) inherited from a diseased mother plant and present in the growing bean plant. This view is supported by the observations of several investigators, who found that the disease can be carried in the seed and that neighbouring plants of known susceptible varieties are attacked to varying extents.

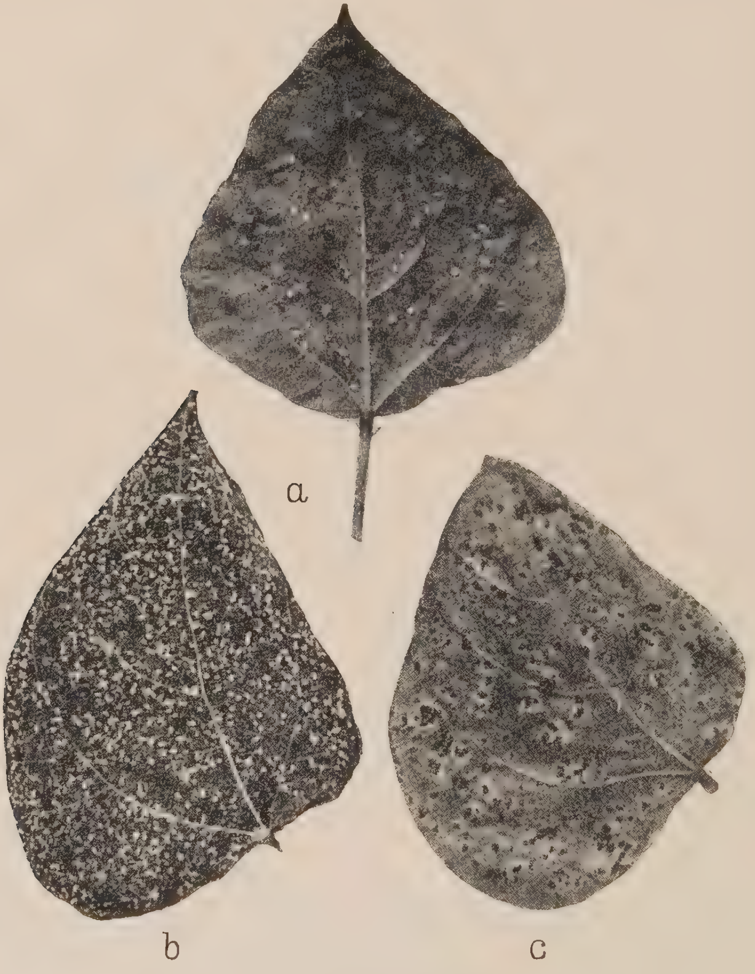

Fig. 136.—Bean Rust.

a, æcidial stage ; *b*, uredo-stage ; *c*, teleuto-stage. (Orig.)

Thus it follows that the spread of the disease by external infection is of much less importance than infection from within.

The disease is widespread ; in severe cases it destroys all the leaves and prevents the formation of pods and seeds. Seed obtained from healthy stocks is the best means of prevention.

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Uromyces Vignæ Fromm., also named *Nigredo Vignæ*, on *Vigna sinensis*, *V. repens*, etc., is closely related to the above fungus; it is found in America (Fromme, 1924).

Uromyces caryophyllinus (Schr.) Wint. produces round or oblong uredo- and teleuto-sori on the leaves and stems of numerous members of the family *Caryophyllaceæ* such as *Dianthus caryophyllus*, *D. barbatus*, *Tunica prolifera*, *Saponaria ocymoides*, *Gypsophila paniculata*, *Arenaria graminis*, etc. The forms on *Tunica prolifera* and *Saponaria ocymoides* have been shown to have an æcidial stage on *Euphorbia Gerardiana*. Inoculation experiments carried out in 1913 in Switzerland, show that the habitat can exercise an influence upon the biological nature of the fungus. Thus a strain of the fungus collected at Wallis (Switzerland) was transmitted equally well to *Saponaria* and *Tunica*, whereas a strain from Heidelberg (Baden) infected *Tunica* almost solely. This difference is said to be due to the fact that in Wallis both host plants are common, whilst throughout Baden, *Saponaria* was not to be found (Fischer, 1913). In America the uredospores were found to be viable at the end of 185 days (Maneval, 1924). Since the end of last century, carnation growing in America, both under glass and out-of-doors, has been seriously affected owing to the attacks of this fungus.

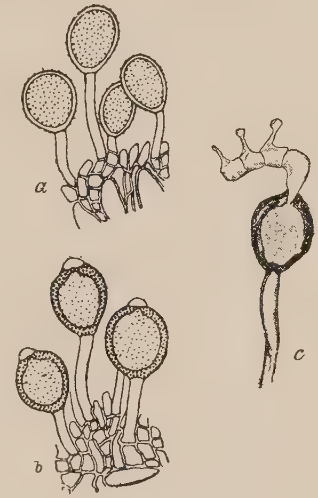

Fig. 137.—Bean Rust. *a*, uredospores; *b*, teleutospores; *c*, teleutospore, after germination. (*a* and *b*, after E. J. Butler; *c*, after E. Prillieux.)

Control Measures:

(1) Resistant sorts of carnations should be selected. (2) Wire netting should be placed between the rows of plants to keep the foliage from the damp ground and to allow of the plants being easily watered when required (Duggar, 1909).

Uromyces Aconiti-Lycoctoni (DC.) Wint. gives rise at first to yellowish groups of æcidia and later brownish-black powdery teleuto-sori on the leaves of *Aconitum Lycoctonum*. The fungus does not infect *Aconitum Napellus*, *A. paniculatum*, and *Trollius europæus* (Jacky, 1899). **U. ambiguus** (DC.) Lév. produces on both sides of the leaves of *Allium rotundum*, *A. Scorodoprasum*, *A. Schœnoprasum*, etc., scattered, rust-coloured uredo-sori and afterwards elongated, often coalescent, chestnut-brown teleuto-sori which remain for a long time covered by the leaden-grey epidermis.

Uromyces Scillarum (Grev.) Wint. appears on pale, round spots on the leaves of *Muscari comosum*, *M. racemosum*, *M. tenniflorum*, *Scilla bifolia*, and *Sc. umbellata* as grouped, coalescent, dark brown, powdery teleuto-sori which are partly covered by the ruptured epidermis (Bubák, 1902). In Switzerland the form on *Muscari racemosum* could not be transmitted to *M. botryoides*. The infection of the host plant takes place in the autumn of the year in which the spores are produced or in the following spring (Schneider, 1912). Closely related forms are: **U. Gageæ** Beck. with dark brown teleuto-sori and smooth spores, found on *Gagea lutea*; and **U. Ornithogali** Lév. with brownish-black teleuto-sori and verrucose spores, found on *Gagea saxatilis* (Bubák, 1902). **U. Lilii** (Lk.) Fuck. appears on both sides of the leaf as well as on the leafstalks and stems of *Lilium bulbiferum*, *L. candidum*, *Fritillaria Meleagris*, etc. At first, pycnidia and æcidia are produced, which project from a raised epidermal pustule; later, brown, powdery teleuto-sori. **Uromyces Primulæ** Fuck. on the leaves of *Primula hirsuta* and *P. viscosa* and **U. Auricolæ** (Magn.) Buchh. on the leaves of *Primula auricola* and *P. pubescens* produce at first scattered æcidia and later also (uredo- and) teleuto-sori. Very closely related is **U. integrifoliae** DC. on *Primula integrifolia*. In all these forms the fungus appears to live perennially in the root stock (Buchheim, 1924). **U. phyteumatum** (DC.) Ung. develops small, dark brown, closely grouped teleuto-sori on the under side of the leaf of *Phyteuma betonicifolium*, *P. Halleri*, etc. **U. Valerianæ** (Schum.) Fuck. forms irregularly, or annularly, arranged æcidia and scattered uredo- and teleuto-sori; found on a number of species of *Valeriana*. **U. verruculosus** Schröt appears as light brown uredo-sori and brownish-black, annulate teleuto-sori on the under side of the leaves of species of *Melandrium*, *Silene*, and *Cucubalus*.

Uromyces Genistæ-tinctoriæ (Pers.) Wint. forms brown uredo- and teleuto-sori on the under side of the leaves of *Genista tinctoria*. The fungus develops an æcidial stage on *Euphorbia Cyparissias* (Fischer, 1920). Similar forms of *Uromyces* are also found on different species of the genera *Cytisus*, *Sarothamnus*, *Colutea*, and *Caragana*. **U. Glycyrrhizæ** (Rabh.) Magn. develops closely and regularly distributed uredo- and teleuto-sori on the under side of the leaves of *Glycyrrhiza glabra*, *G. hirsuta*, etc. The fungus appears to hibernate in the root stock of the host plant (Magnus, 1890). **U. Veratri** (DC.) Schröt. appears on the lower surface of the leaves of *Veratrum album* and *V. nigrum* as yellowish-brown uredo-sori and chestnut-coloured teleuto-pustules. The fungus develops pycnidia and æcidia on *Adenostyles alpina* (f. sp. Adenostylis) and *Homogyne alpina* (f. sp. Homogynes) (Fischer, 1908; Tranzschel, 1915). **U. lupinicolus** Bub. produces cinnamon-brown uredo- and blackish-brown teleuto-sori on the leaves of *Lupinus angustifolius*.

Two different species of *Uromyces* are found on the genus *Alchemilla*. The first, **Uromyces Alchemillæ** (Pers.) Wint., breaks out in spring on the under surface of certain leaves of *Alchemilla vulgaris*. These leaves have abnormally long stalks and a stunted blade and on them closely-grouped, yellow uredo-sori are produced; later a few teleutospores appear amongst the sori. The other species **U. melosporus** (Thorry) Syd. develops closely-grouped, brown teleuto-sori on the under surface of the leaves of *Alchemilla alpina* (subsp. *Hoppeana*); a few uredospores are intermingled with the teleuto-sori. Both species appear to hibernate in the root stock (Fischer, 1915).

Uromyces Geranii (DC.) Wint. produces æcidia on the thick portions of the leaves of a number of species of *Geranium* such as *Geranium silvaticum*, *G. Phœum*, *G. dissectum*, *G. molle*, *G. pyrenaicum*, etc., while round uredo- and teleuto-sori appear on the under surface of the leaves. Another species, **U. Kabatanus** Bub., is found on *G. pyrenaicum* and *G. dissectum*. Specialization does not, however, seem to be extensive in the species (Bock, 1908). **U. Solidaginis** Som. produces brownish-black teleuto-sori surrounded by a yellow halo on the blades and stalks of leaves of *Solidago Virgaurea* (also on *S. canadensis* in America). The fungus has no other stage of development (Fischer, 1905).

A number of autœcious *Uromyces* attack Euphorbia plants. **Uromyces scutellatus** (Schrenk.) Lév. gives rise at first to pycnidia, and sometimes also to æcidia, on the under surface of the leaves of *Euphorbia Cyparissias*, *E. Gerardiana*, *E. Esula*, *E. verrucosa*, *E. lucida*, etc.; later a teleuto-stage appears on the under surface of the stunted leaves. The sori, when ripe, are covered with raised portions of the leaf tissue, and in some cases there is an intermingling of uredospores. The fungus lives perennially in the host plant and causes deformation of the shoots. Closely related to the latter is **U. excavatus** (DC.) Lév. which has all its spore forms on the under side of the leaves of *Euphorbia verrucosa* and *E. dulcis*. In each species it is probably a case of a specialised form (Tranzschel, 1910). **U. tuberculatus** (Fuck.) Wint. develops uredo- and teleuto-sori on the leaves and stalks of *Euphorbia exigua* without causing deformation of the organs.

Gymnosporangium

The teleutospores are two-celled and embedded in a mucilaginous matrix, and are provided with stalks that swell considerably. Some of the spores are thin-walled and others thick-walled; the latter germinate by a short promycelium from which sporidia arise laterally. The thin-walled spores germinate by elongated, straight hyphæ from the tips of which conidia are abstricted.

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Juniper Rust

Gymnosporangium Sabinæ (Dicks.) Wint.

The internal mycelium of this fungus forms long, spool-like thickenings on the stems and twigs of the juniper tree (*Juniperus Sabina*). In April or May crowded, brown, becoming brownish-yellow, cone-shaped teleutospores with rounded tips arise. During dry weather these lesions are shrivelled and hardly noticeable, but in a moist atmosphere, especially after rain, they swell up and become mucilaginous. Some of the teleutospores are thick-walled, others thin-walled, and they are able to germinate at once. As a result of infection by germinating teleutospores, sticky patches with black dots (pycnidia) are produced on the upper surface of the leaves of the pear (*Pyrus communis*) and soon afterwards yellowish-red to brown groups of æcidia appear on the under side of these patches, as well as on the young pear fruits. The cup of the æcidium does not open completely and the tips form a sort of grating. The infected fruits are misshapen and inedible.

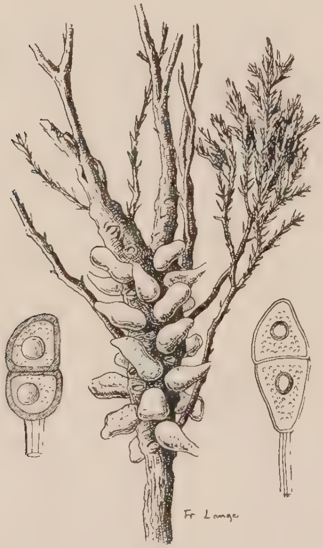

Fig. 138.—Juniper Rust.

Twig with teleuto-masses. Left a thick-walled, right a thin-walled teleutospore. (After E. Rostrup.)

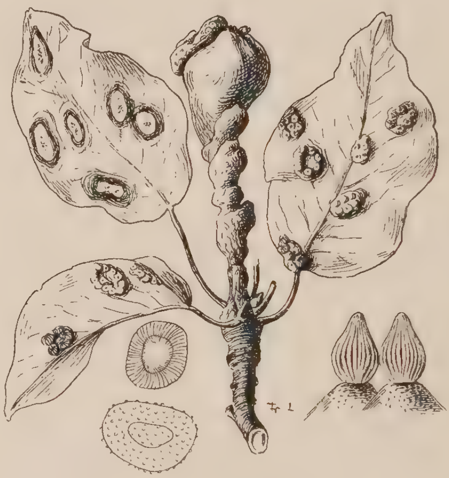

Fig. 139.—Juniper Rust.

Leaves and fruit of pear with æcidia. Left, æcidiospores, one in section; right, æcidial cups. (After E. Rostrup.)

The teleuto-stage of the fungus (on the Juniper) was first observed in England in 1785 and described under the name *Tremella Sabinæ* Dicks. Some years later (1791) the æcidial stage was described as *Æcidium cancellatum*. In 1837, the injury caused to pear trees by the juniper rust was noticed and reported upon. The genetic relationship of the two forms of the fungus was first proved in 1865, as a result of inoculation experiments made in Denmark (Oersted).

In certain cases it has been shown that the mycelium can continue to live on the pear tree without change of host. Thus in a glasshouse in Munich in the spring of 1907, young pear trees, which had been diseased in the previous year and had been kept in the house, produced normal æcidia in large numbers (v. Tubeuf). In Northern Caucasia, pear rust was severe in the open, even in places where no junipers were to be found in the vicinity (Andreyeff, 1924). Reports also show that in spite of the extermination of the juniper, serious outbreaks of disease have recently occurred in Denmark (Gram and S. Rostrup, 1922) and in Italy (Voglino, 1923). The fungus does not appear to develop on the juniper further north than Denmark and the south of Sweden.

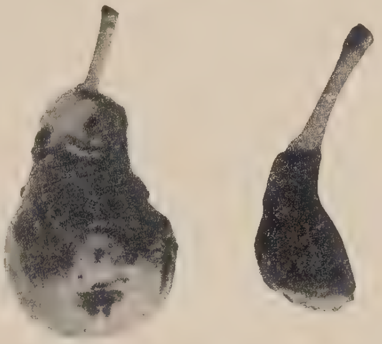

FIG. 140.—Misshapen Pears covered with æcidia. (Orig.)

As a protective measure it is recommended to exterminate all infected juniper trees in the neighbourhood of pear orchards.

The two following species of this type of rust are very closely related to one another and are probably best regarded as specialized forms; they both occur on *Juniperus Sabina:* **Gymnosporangium confusum** Plowr. with the æcidial stage (*Æcidium Mespili*) on *Mespilus germanica*, *Cratægus Oxyacantha*, *C. monogyna*, *Cydonia vulgaris*, *C. oblonga*, *Sorbus torminalis* and *S. latifolia*, this form has been observed in England, Germany, Austria, Switzerland, etc. (=*G. Sabinæ* f. sp. *Mespili*) (Plowright, 1884, 1888; Fischer, 1891, 1898, 1904; Klebahn, 1892, 1904) and **G. fusisporum** Fisch. with æcidial stage (*Æcidium Cotoneasteris*) on *Cotoneaster vulgaris* and very feebly on *Pyrus communis;* this form has been observed in Switzerland (=*G. Sabinæ* f. sp. *Cotoneastris*) (Fischer, 1917).

In Sweden, especially in the south, outbreaks of disease have occurred in a number of places resulting in the pears being misshapen and more or less covered with æcidia; in no case could the disease be attributed to the proximity of *Juniperus Sabina*. In some instances there appeared to be a connection between the outbreaks of disease and neighbouring bushes of *Juniperus communis*, in which case the fungus *Gymnosporangium clavariæforme* f. sp. *Pyri-communis* would come into account. As, however, the æcidial form on the pear appears each year on the same trees and not on adjacent ones, it must be concluded that this æcidium can over-winter on the pear tree when conditions are favourable.

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Juniper "Cushion" Rust

Gymnosporangium tremelloides (Al. Br.) v. Tub.

The teleuto-sori of this fungus break out on the common juniper (*Juniperus communis*) from April to May, partly as broad, almost shell-shaped, chocolate-brown growths on the stem, the spore contents becoming exposed by the bursting of the epidermal tissue, and partly as small, wart-shaped, chocolate-brown cushions on the leaves. After rain the fructifications swell and produce noticeable masses of yellow mucilage. Two different forms of teleutospores are found in the fructifications: the peripheral ones are of a dark brown colour and thick-walled, with the component cells firmly joined; the central ones are thin-walled, of a light brown colour, and the component cells loosely joined so that they separate easily.

The thick-walled cells germinate by a short, broad, slightly-bent promycelium from which sporidia are abstricted on the outer side. On the other hand, the thin-walled spores germinate by long, slender tubes which separate conidia at their tips. To what extent the two spore forms have a different function in the life of the fungus is not yet known.

The fungus develops æcidia on a number of *Pomaceæ*. At least five different specialized forms are distinguished: (1) f. sp. Aucupariæ with the æcidial stage (*Rœstelia cornuta*) on *Sorbus Aucuparia* (Denmark,

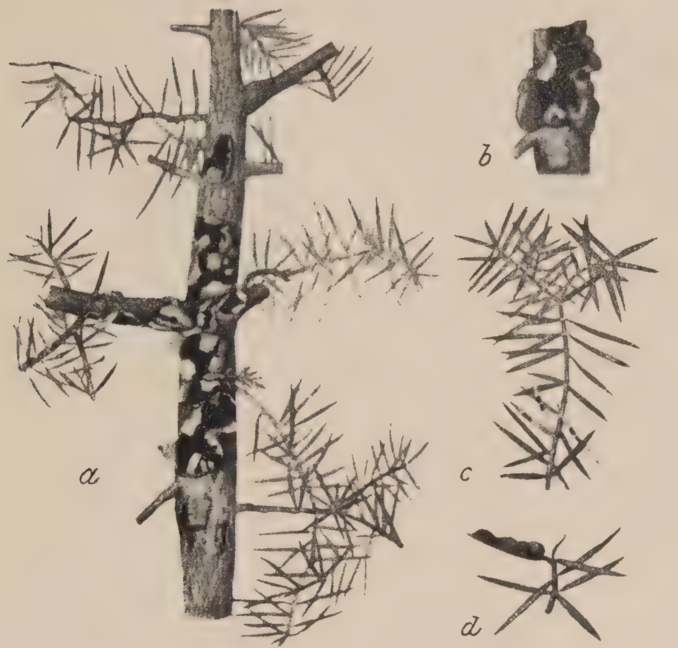

FIG. 141.—Juniper "Cushion" Rust.

a, dry cushions of fungus on stem; *b*, fungus cushion swelling up; *c*, dry cushions on leaves; *d*, swollen leaf cushion. (Orig.)

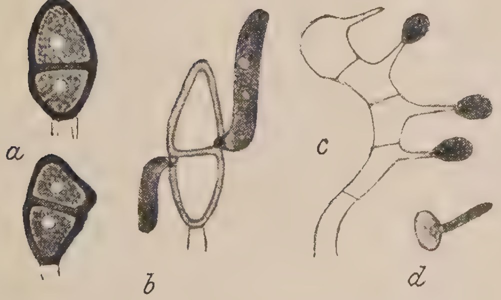

FIG. 142.—Juniper "Cushion" Rust.

a, thick-walled spores; *b*, thick-walled spore after germination, beginning of promycelium; *c*, promycelium with sporidia; *d*, germinated sporidium. (Orig.)

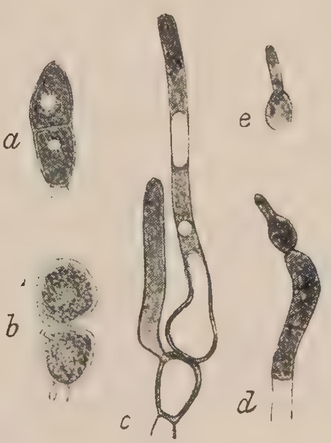

FIG. 143.—Juniper "Cushion" Rust.

a and *b*, thin-walled spores; *c*, germinated thin-walled spore; *d*, tip of hypha abstricting conidium; *e*, germinated conidium. (Orig.)

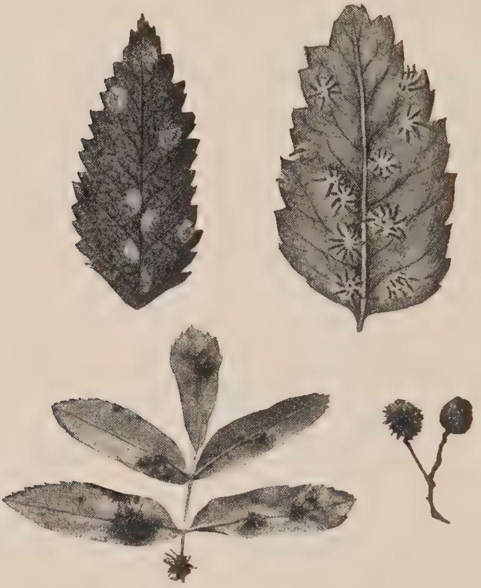

Fig. 144.—Juniper "Cushion" Rust.

Æcidial stage on mountain ash (f. sp. Aucupariæ). Above, two leaflets; upper surface shown on left, lower surface on right. Below, part of leaf and fruits attacked by *Rœstelia cornuta*. (Orig.)

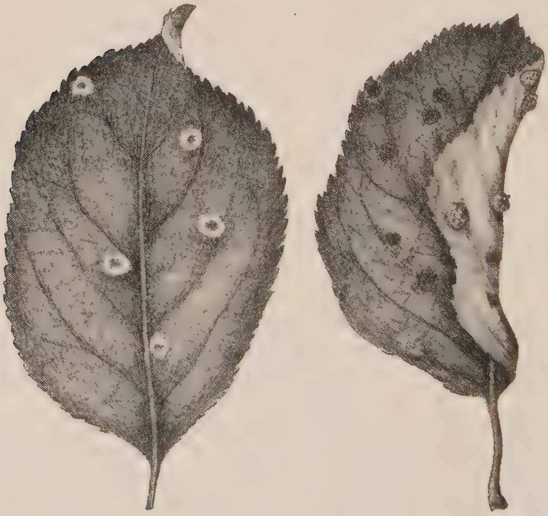

Fig. 145.—Juniper "Cushion" Rust.

Æcidial stage on apple (f. sp. Mali). On left, leaf with patches of spermogonia; on right, leaf with patches of æcidia (*Rœstelia penicellata*). (Orig.)

Germany, Switzerland, Sweden); (2) f. sp. Mali with the æcidial stage (*Rœstelia penicellata*) on *Pyrus Malus* (Denmark, Russia, Sweden); (3) f. sp. Amelanchieris with the æcidial stage (*Rœstelia Amelanchieris*) on *Amelanchier vulgaris* (Germany, Switzerland); (4) f. sp. Torminalis with the æcidial stage on *Sorbus torminalis* and *S. latifolia* (Switzerland); (5) f. sp. Ariæ with the æcidial stage on *Sorbus Aria* (Germany, Switzerland).

The only form that has economic importance is the one that produces æcidia on the leaves of the apple tree. The frequent appearance of *Rœstelia penicellata* on apple leaves (for example, near Stockholm) in districts where the complimentary host—the juniper—is rare or separated by considerable distance, as well as the low infective power of the fungus makes it difficult to explain the outbreaks. The question therefore arises whether the fungus is able to over-winter on the tree, perhaps in the buds.

The teleuto-stage of the fungus was first mentioned under the name of *Byssus gelatinosus* in 1737 and in 1753 as *Tremella juniperina* Lin. The æcidial stage on *Sorbus Aucuparia* was distinguished in 1791 as *Æcidium penicellatum*, and on *Pyrus Malus* in 1803 as *Æcidium Mali* Schum. The genetic relationship between the teleuto- and æcidial-stages was first shown experimentally in Denmark in 1866 (Oersted).

The removal of junipers from the vicinity of a fruit garden is regarded as the best means of controlling the disease.

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Juniper "Cone" Rust

Gymnosporangium clavariæforme (Jacq.) DC.

The teleuto-sori of this fungus break out on the thickened parts of the stem and branches of the common juniper, in the form of long, cylindrical, bright-yellow cones or tongues which swell up after rain. The fructifications contain two different forms of teleutospores: in the peripheral parts of the sori the spores are deep brown and thick-walled, the component cells adhere closely; in the centre the spores are light brown, thin-walled, with component cells that are narrow and do not adhere closely.

The thick-walled spores germinate by a short, slightly-bent promycelium which produces sporidia at the sides; the thin-walled spores germinate by long tubes from the tips of which conidia are abstricted.

The fungus develops its æcidia on a number of *Pomaceæ*. Up to the present, at least three specialized forms have been distinguished: (1) f. sp. Cratægi with the æcidial stage (*Æcidium laceratum*) on *Cratægus coccinea*, *C. Douglasii*, *C. grandiflora*, *C. monogyna*, *C. nigra*, *C. Oxyacantha*, *C. punctata*, *C. sanguinea*, and *C. tomentosa* (Austria, Germany, Switzerland, Sweden, England, and America); (2) f. sp. Pyri communis with the æcidial stage on *Pyrus communis* (Austria, Germany, Switzerland, England); (3) f. sp. Amelanchieris with the æcidial stage (*Æcidium Amelanchieris*) on *Amelanchier alnifolia*, *A. canadensis*, *A. erecta*, *A. intermedia*, *A. oblongifolia*, *A. oreophila*, *A. polycarpa*, *A. pumila*, *A. vulgaris* (=*Gymnosporangium Amelanchieris* Fisch) (Germany, America).

In Italy (Pavia) the æcidial stage of the fungus has been met with each year on certain shrubs of *Cratægus Oxyacantha* although junipers were not to be found in the vicinity, at least not within many miles. It has consequently been thought that the mycelium of the æcidial stage over-winters on *Cratægus* (Montemartini, 1925).

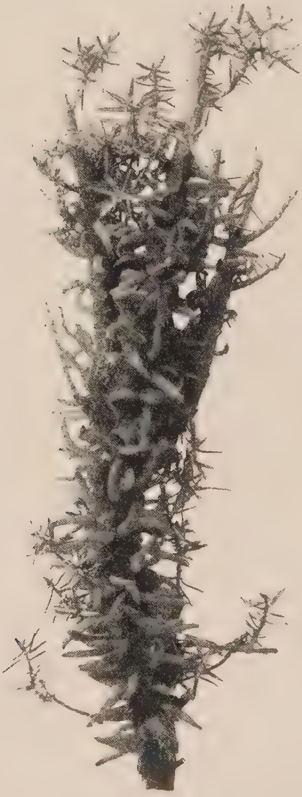

Fig. 146.—Juniper "Cone" Rust. "Cones" of fungus in dry state. (Orig.)

This fungus has been known in its teleuto-stage on juniper as *Tremella clavariæformis* Jacq. since 1788 and in its æcidial stage on *Cratægus Oxyacantha* as *Æcidium laceratum* Sow. since 1801, the genetic relationship of the two stages of development was proved experimentally in 1867 (Oersted).

In the United States it has been possible to transmit the fungus from *Juniperus sibirica* to *Amelanchier erecta*, *A. intermedia*, *A. canadensis*, *Cratægus tomentosa*, and *C. punctata*.

To control the disease, juniper bushes in the vicinity of the fruit garden must be removed.

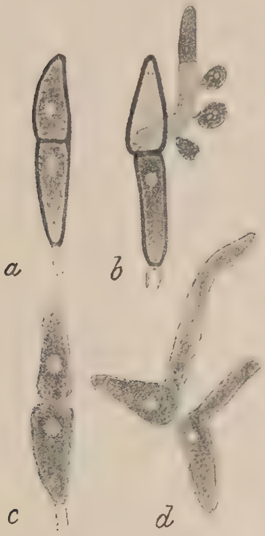

Fig. 147.—Juniper "Cone" Rust.

a and *b*, thick-walled spores; *b*, germinated with promycelium; *c* and *d*, thin-walled spores; *d*, germinated with germ-tubes. (Orig.)

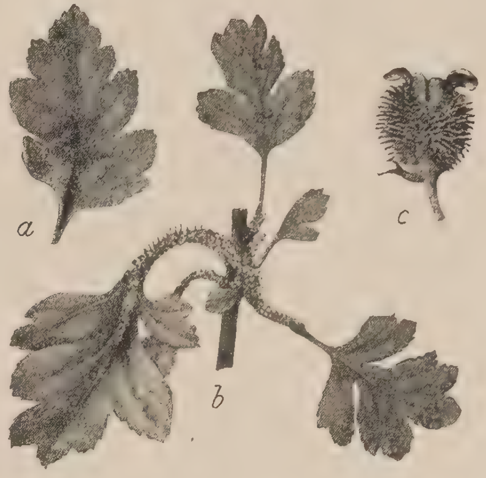

Fig. 148.—Juniper "Cone" Rust.

Æcidial stage on Thorn (f. sp. Cratægi). *a*, leaf; *b*, leaf-stalk; *c*, fruit attacked by *Rœstelia Oxyacanthæ*. (Orig.)

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In Southern Russia the following are known: **Gymnosporangium Oxycedri** Bres. on *Juniperus Oxycedrus*, with æcidia on *Cratægus monogyna*, *C. nigra*, *Mespilus germanica* and *Sorbus torminalis*; and **G. tauricum** Eriks. also on *Juniperus Oxycedrus* with æcidia on *Cratægus monogyna* (Eriksson, 1919).

Gymnoconia Peckiana (Howe) Tranz. occurs on a number of cultivated and wild species of the genus *Rubus*. The spermogonia are the first to develop on the leaves in spring; they cover the whole or part of the leaf on both sides with a bright yellow, verrucose covering. In certain cases, as with *Rubus saxatilis* and *R. arcticus*, the infected shoots shrivel up. The cæoma stage (*Cæoma interstitiale* Schlecht., *C. nitens* Brur.), which appears somewhat later, is restricted to the under surface of the leaf, and when the disease has reached this point the leaves gradually die off. As the fungus hibernates in the stem, an infected plant produces new cæoma pustules each year and finally dies. The last stage of development of the fungus—the teleuto-stage—only occurs to a limited extent and in certain countries (such as Northern Sweden and Northern Russia). Small brownish-black pustules are produced on the under surface of the leaf. Only the pycnidial and cæoma stages are of economic importance and they often cause considerable damage in America; they have been met with in a number of European countries (Bavaria, France, Finland, Russia). In certain cases the pycnidial stage does not develop. Cultivated varieties differ in the degree to which they suffer from the disease.

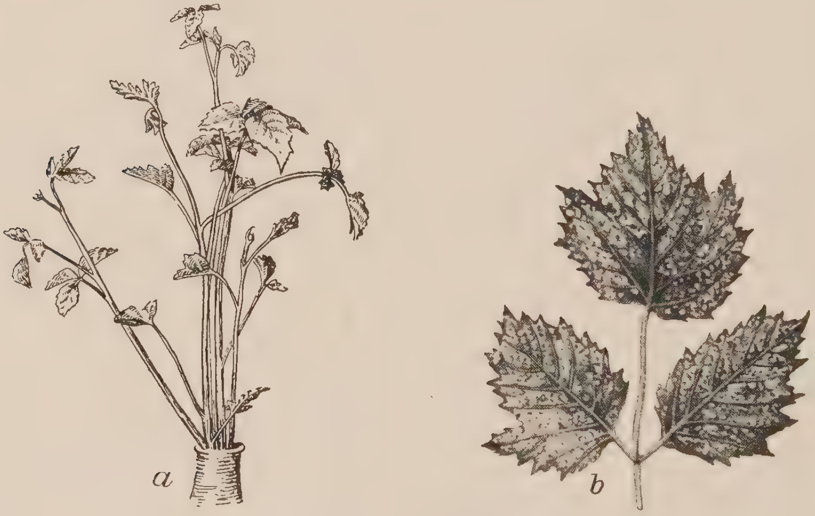

Fig. 149.—*Gymnoconia Peckiana.*

a, blackberry shoot deformed by attack of fungus; *b*, leaf, covered below with cæoma pustules. (*a*, after B. M. Duggar; *b*, after L. O. Kunkel.)

It has been recommended in America that to control the disease all new plantations should be carefully inspected not later than a month after planting and all infected plants removed (Tranzschel, 1893; Kunkel, 1916, 1920; Arthur, 1917; Stone, 1918; Dodge, 1923; Cunningham, 1924).

Phragmidium

The teleutospores are two- to many-celled with the cells superimposed.

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Rose Rust

Phragmidium subcorticium (Schr.) Wint.

The cæoma stage of this fungus is found on the under surface of the leaves as well as on the petioles, stems, and sepals, even on the fruits (hips) of a number of varieties of roses such as *Rosa centifolia*, *R. cinnamomea*, *R. gallica*, *R. rubrifolia*, *R. canina*, etc. The disease appears in spring in the form of swollen, often elongated, orange-yellow cushions. The cushions on the leaf veins may have a length of 5 mm. and those

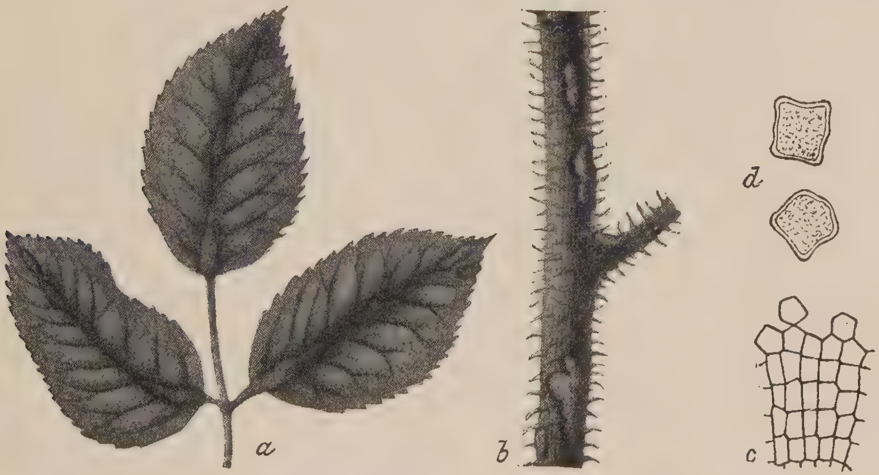

FIG. 150.—Rose Rust, Cæoma Stage.

a, leaflets; *b*, stem attacked by fungus; *c*, abstriction of cæomaspores; *d*, cæomaspores. (Orig.)

on the stem 10 mm. The affected parts often hypertrophy and become twisted and bent. A pycnidial stage of the fungus precedes the cæoma stage on the blade of the leaf.

At the end of June pale, orange-yellow, circular uredo-sori appear on the under surface of the leaf, and at the end of July, if not earlier, this surface is thickly covered with black teleuto-pustules. The infected leaves turn yellow and fall prematurely, the flowers either develop badly or not at all.

Sometimes, and especially in the case of roses such as *Rosa rubrifolia*, the cæoma stage is very severe and injurious. In such cases the hymenium in the stem can live on for three successive years and repeatedly produce spores; no uredo- or teleuto-stage occurs, and the mycelium hibernates in the primary cortex of the stem. During the winter the hymenium, which produces the spores, is covered by the bark. When the pustules open, a mass of rust-red spores is liberated. Badly infected rose trees often die completely in the first year of the disease. Inoculation experiments with cæoma-spores which had been produced in this way gave negative results even when the spores had a very high germinating power.

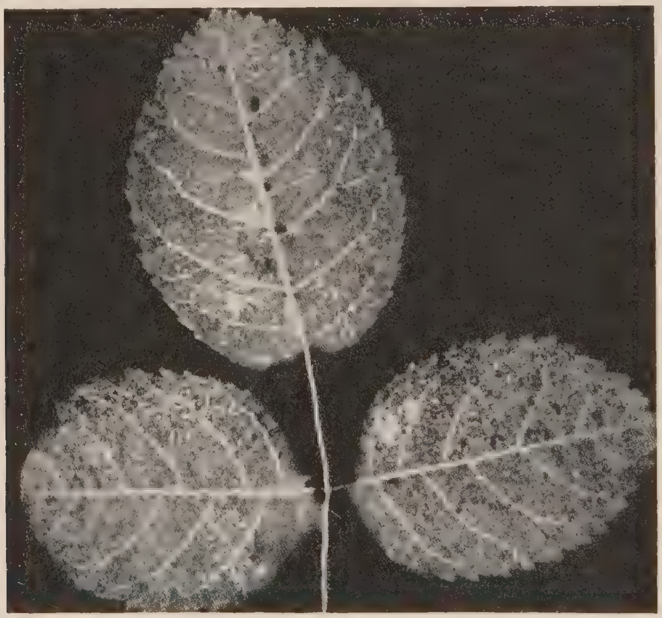

Fig. 151.—Rose Rust, Uredo-Stage. (Orig.)

In most cases the fungus over-winters in the usual way by means of teleutospores which, when kept in the open, produce the various spore stages in the course of the following year.

Within this species, two well-specialised forms have been distinguished in Switzerland and in Sweden: (1) f. sp. centifoliæ on *Rosa centifolia*, *R. gallica*, *R. hybrida*, and *R. canina;* and (2) f. sp. cinnamomeæ on *Rosa cinnamomea*, *R. pimpinellifolia*, and *R. rubrifolia.*

Garden roses differ in their susceptibility to rust, the so-called remontant or perpetual roses suffering severely, most of the tea roses much less, whilst the climbing and polyanthus roses are almost immune.

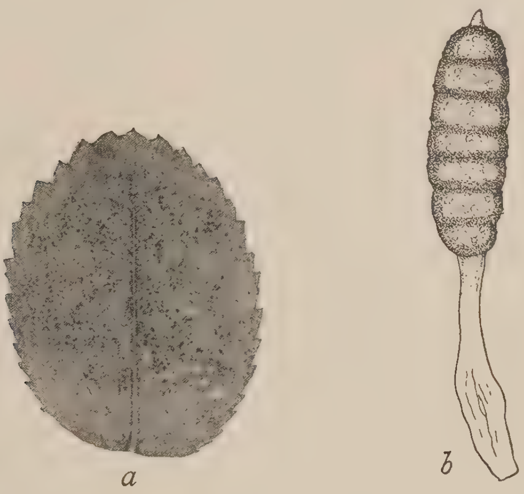

FIG. 152.—Rose Rust, Teleuto Stage.
a, leaflet with teleuto-pustules on under surface; *b*, teleutospore. (Orig.)

To control the disease, it is recommended to collect and destroy all rusted leaves in autumn and to spray the heads as well as the stems with Bordeaux mixture. Further, to cut out, in spring, and destroy all parts of the plant bearing the cæoma stage and to paint the infected parts of the stem with carbolineum.

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The following species are related: **Phragmidium tuberculatum** J. Müll. with purple-red spots on the upper surface of the leaves of *Rosa cinnamomea*, *R. canina*, *R. rubrifolia*, etc. On the under surface of the leaf are uredospores with a thick, echinulate membrane. **P. fusiforme** Schröt, with pycnidia- and cæoma-pustules of different sizes on all the green parts of the plant, and with pustules of uredo- and teleutospores on the under surface of the leaves of *Rosa alpina.*

Phragmidium Rubi-Idæi (DC.) Karst. Raspberry Rust. The cæoma stage of this fungus appears on the upper surface of the leaves of *Rubus Idæus* about the middle of June. This stage is characterized by small, yellow, circular, isolated pustules and by large, yellow patches on the sepals of the flower. About three weeks later, the uredo generation appears in the form of small, bright yellow pustules regularly distributed over the under surface of the leaf. Finally, about the end of July, the teleuto generation appears also on the under surface of the leaf; the sori are black and powdery and lie near to and between the uredo-pustules. Severely infected leaves soon wither and drop prematurely. The teleutospores hibernate in the open and germinate in spring, and the sporidia which arise therefrom produce a more or less severe eruption of cæoma pustules. Inoculation experiments, carried out in 1901 near Stockholm, with cæoma- and uredospores yielded negative results, the observations extending to a period of 15–33 days (Eriksson, 1923).

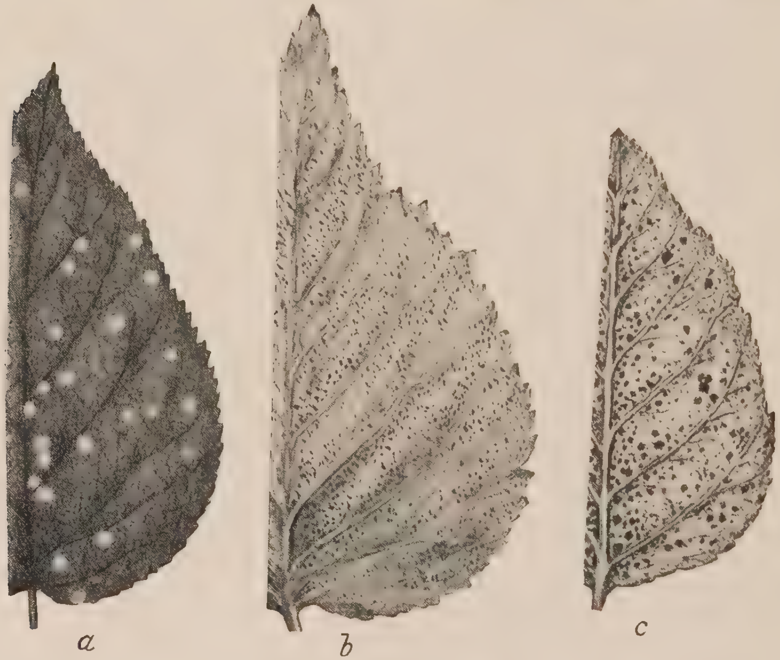

Fig. 153.—*Phragmidium Rubi-Idæi.*

Raspberry leaves: *a*, with cæoma-pustules on upper surface; *b*, with uredo-pustules on under surface; and *c*, with teleuto-pustules on under surface. (Orig.)

Phragmidium violaceum (Schultz) Wint. Blackberry Rust. This fungus produces pycnidia on the upper surface of the leaves of a number of different sorts of blackberries such as *Rubus fruticosus*, *R. laciniatus*, *R. thyrsoideus*, etc. The succeeding spore-generations are on the under surface of the leaf in the form of violet-red spots. The power of the fungus to produce infection in artificial cultures as well as its capacity to spread in the open, appears to be astonishingly limited (Eriksson, 1923).

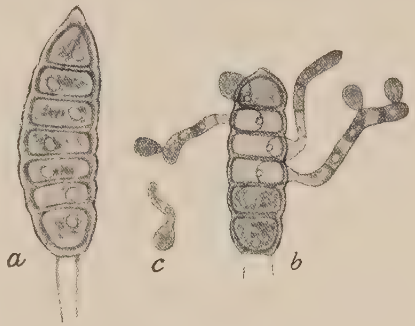

Fig. 154.—*Phragmidium Rubi-Idæi.* *a*, teleutospore; *b*, germinated teleutospore; *c*, sporidium, germinated. (Orig.)

Phragmidium Potentillæ (Pers.) Karst. attacks many species of *Potentilla* such as *Potentilla argentea*, *P. nivea*, *P. cataclines*, *P. thuringiaca*, *P. argyrophylla*, etc. The fungus gives rise to a small number of pycnidia- and cæoma-spots on both sides of the leaf. Shortly after these appear, crowded brownish - yellow uredo-sori and black teleuto-sori are found on the under surface of the leaf. The capacity of the fungus to spread seems at times to be very limited. Thus it was observed in 1900–03, in the vicinity of Stockholm, that a plot of *Potentilla cataclines* suffered severely each summer and autumn through the uredo- and teleuto-generations, whilst other species of *Potentilla* close by were entirely free from rust (Eriksson, 1923).

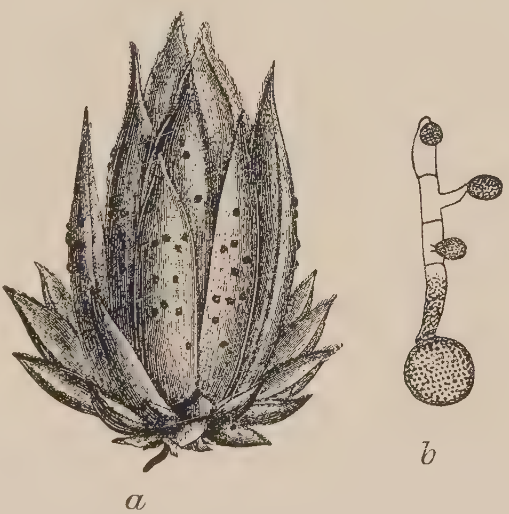

Fig. 155.—*Endophyllum Sempervivi.* *a*, house-leek attacked by fungus; *b*, teleutospore, germinated. (After A. Kerner.)

Endophyllum Sempervivi (Alb. & Schw.) de By. This fungus is found on *Sempervivum tectorum*, *S. montanum*, *S. arachnoideum*, in early spring. Orange-red, globular pycnidia appear and large sunken teleuto-sori which have one-celled teleutospores that germinate and form promycelia. The fungus perennates in the host plant, causing the leaves to become misshapen; the plants are often severely damaged (Werth, 1913).

Chrysomyxa

The teleutospores are one-celled, with a thin colourless membrane, united, in velvety cushions, to simple or branched rows of cells, germinating at once after ripening with formation of promycelium. Æcidiospores and uredospores, when such occur, are produced in chains with paraphyses between.

Chrysomyxa Abietis (Wallr.) Ung. This fungus makes itself apparent in early summer on young fir trees in the form of small bright-yellow spots on the newly developed leaves. Towards the autumn, the spots enlarge, become more oblong, and of a lemon-yellow colour. Several of these pulvinate masses are found on each leaf. The teleutospores are the only spore-generation and they are united in rows—up to twelve in a row—and are covered with a thin colourless membrane; they germinate at once when ripe. After the spore-masses have over-wintered the membrane ruptures in the spring, the spores germinate by promycelia and infect the young shoots which are being formed at the same time. In late summer, the presence of mycelium in the young leaves is indicated by the formation of yellowish rings. The leaves which bear pustules in the spring fall in the autumn.

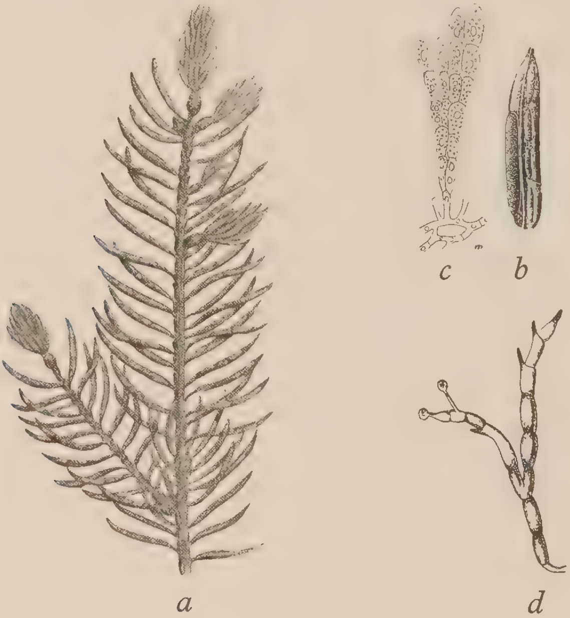

Fig. 156.—*Chrysomyxa Abietis*.

a, branch of fir with rusted needles; *b*, rusted needle; *c* and *d*, teleutospores; *d*, germinated. (*a*, Orig.; *b* and *c*, after E. Rostrup; *d*, after E. Delacroix and A. Maublanc.)

It is usually the young—at most twenty years old—firs that are attacked, and the disease generally returns to the same tree each year. A badly infected tree becomes more and more shrub-like and short of leaves until finally it dies.

Sometimes it may be noticed that alongside a badly diseased tree there are others which are only slightly attacked or not at all. When a tree has been so weakened by the repeated attacks of the fungus that it is likely to die, or when an individual tree is seen to be infected and those around it healthy, it is advisable to destroy such a diseased tree and so check the spread of the disease.

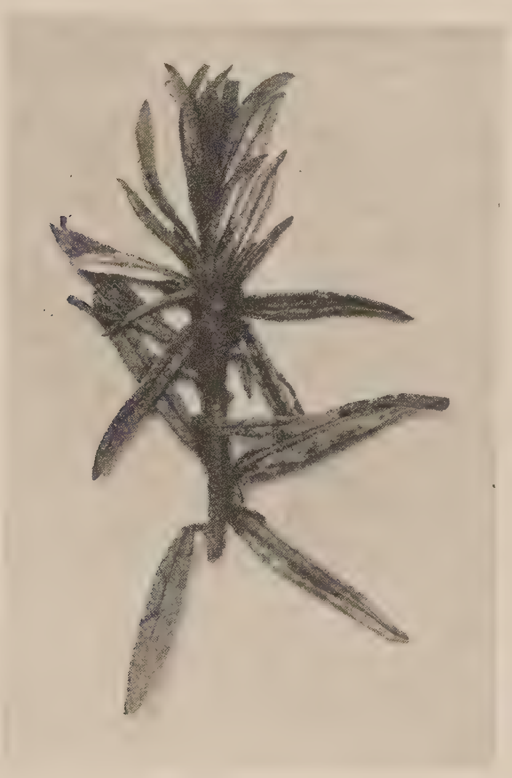

FIG. 157.—*Chrysomyxa Ledi.* Teleuto-stage on the under surface of leaves of marsh rosemary. (Orig.)

In America, a closely related form **Chrysomyxa Weirii** Jacks. has been found on *Picea Engelmannii* (Jackson, 1917; Weir, 1923).

Chrysomyxa Ledi (Alb. & Schw.) de By. This fungus produces, in May or June, closely grouped, yellowish-red teleuto-sori as well as scattered uredo-sori on the under surface of the previous year's leaves of marsh rosemary (*Ledum palustre*). The spore-masses are laid down in the previous autumn, but the spores only develop and ripen in the spring. When the teleutospores germinate they infect the needles which are appearing at the same time from the buds of *Picea excelsa*. At the beginning of July, pycnidia and æcidia are seen to break out; the latter, which formerly were named *Æcidium abietenum* Alb. & Schw., lie in rows on the under side of the needles and appear as small white tubes which burst at the tip and liberate a yellow dust. This spore-dust is sometimes produced and disseminated in such large quantities (especially in Northern Europe) that it rises in a dense, yellowish-red cloud which later settles. The trees in sunny situations suffer most from the attack of the fungus and only the old, crowded parts of the forest remain free. The æcidiospores infect the leaves of *Ledum* which are produced in summer, and so a new cycle of development is begun.

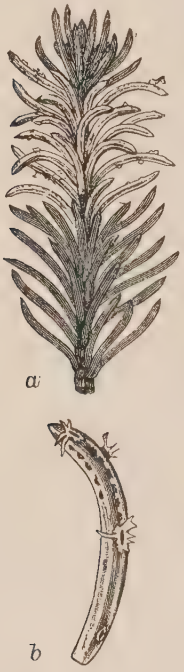

FIG. 158.—*Chrysomyxa Ledi*. Æcidial stage on fir. *a*, shoot with some diseased needles; *b*, single diseased needle. (Orig.)

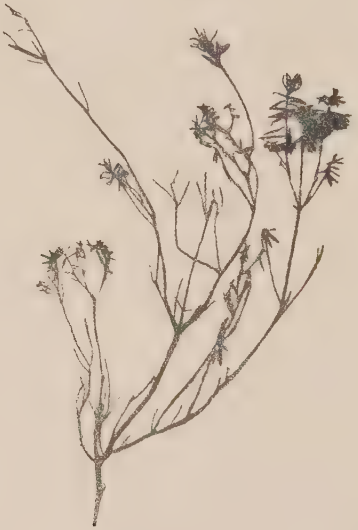

FIG. 159.—*Chrysomyxa Ledi*. Witches' broom on marsh rosemary. (After O. F. Liro.)

By means of the uredo-pustules formed in the previous summer the fungus spreads further to the young leaves and stems and produces secondary uredo-pustules. The mycelium over-winters in the infected organs and for at least two successive summers is able to produce new uredo-pustules; this indicates that the fungus can continue to live on *Ledum* without change of host. Also, at least in northern countries, the mycelium is able to migrate to the new buds and hibernate there. The shoot which arises from such a bud develops abnormally, and when the mycelium remains alive and active in the cortex of the plant for several years it causes a witches' broom to arise. The living terminal shoots, or twigs, are attacked by the fungus and the mycelium can be found in the dead twigs.

Another mode of development of the fungus on the fir is also known and occurs in Northern Europe (Finland, Russia, Sweden). The teleutospores, which are produced on *Ledum* in summer, infect not only the fir leaves that have developed but also, and particularly in certain years, some of the leaf buds, especially the terminal buds. In such cases the mycelium appears to permeate both the leaf- and stem-parts and continues to develop throughout the winter. At the end of April, or the beginning of May, the infection is shown by a slight swelling of the bud which early in June becomes changed to a cone-shaped, deformed, yellowish shoot with short, thick, pale, erect leaves on which golden-yellow æcidia break out. This condition may arise on the lower and middle branches as well as in the head of the tree; generally it is found only on a few trees, but is more common in certain years.

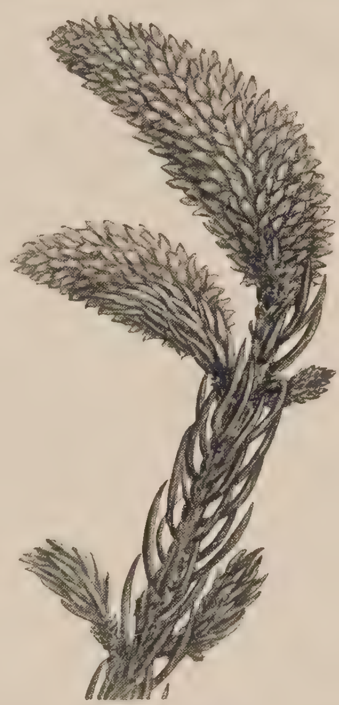

FIG. 160.—*Chrysomyxa Ledi.* Æcidial stage on fir. Cone-shaped, deformed shoot. (*Æcidium corrusans.*) (Orig.)

This æcidium has been known for a long time, the name *Æcidium corruscans* (E. Fries, 1824) having been given to it. At the beginning of the present century, after tender *Ledum* leaves had been inoculated with æcidiospores and teleuto-sori produced, the æcidium was regarded as the æcidial stage of a new *Ledum* fungus *Chrysomyxa Woronini* (Tranzschel, 1903). This new species was considered to be characterized by the presence of teleuto-sori on the new leaves produced by the buds in spring, and not on those of the previous year. A few years later, however, it was finally proved that *C. Woronini* is only a specially developed form of *C. Ledi* and the new species had to be withdrawn (Liro, 1907).

It may be mentioned that the æcidial stage of the fungus is also met with in districts where *Ledum* does not grow.

Chrysomyxa Rhododendri de By. is closely related to *C. Ledi.* The æcidial stage appears on the under side of the needles of *Picea excelsa*, the uredo- and teleuto-stages on the under surface of the leaves of some species of *Rhcdodendron* such as *Rh. ferrugineum* and *Rh. hirsutum.* The fungus is common in mountainous districts but apparently only the uredo-stage appears in glasshouses and botanical gardens of the lowlands (v. Tubeuf, 1893; Lagerheim, 1894; Klebahn, 1905).

Currant Felt Rust

Cronartium ribicola Dietr.

Peridermium Strobi Kleb.

This fungus attacks a number of species of the genus *Ribes*, particularly *Ribes nigrum*, *R. Cynosbati* and *R. americanum*, to a less degree *R. sanguineum*, *R. rubrum*, *R. Grossularia*, *R. Gordonianum*, etc. Small ochre-yellow, warty swellings are produced in summer on the under surface of the leaf. From these swellings uredospores are extruded through a small hole at the apex. One to two weeks later teleuto-sori arise. They are yellowish-brown, cylindrical, raised, slightly curved and when dry almost horny; the spores are arranged longitudinally in rows, side by side and superimposed. The teleuto-sori are at first in groups between the leaf veins, but they soon coalesce and cover the whole of the lower surface with a brown, felt-like coating. The teleutospores are not spread by wind, but they stick together and form small columns which finally cause the under surface of the leaf to become black. The spores are able to

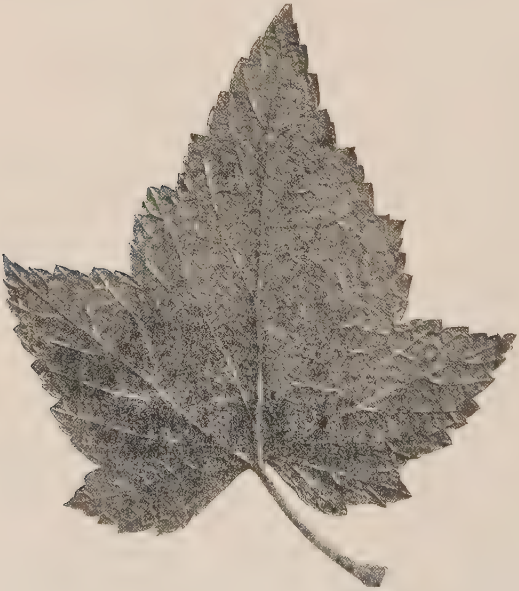

Fig. 161.—Currant Felt Rust. Leaf seen from below. (Orig.)

germinate at once when ripe. The individual spores in the small columns send out a promycelium which produces sporidia.

In this stage—*Cronartium ribicola*—the fungus has been known since the middle of the last century (Dietrich, 1856). It had been observed from time to time seventeen years previously on *Ribes nigrum*, *R. rubrum*, and *R. aureum* in gardens; later it was reported from Denmark (Rostrup, 1871), Germany (P. Magnus, 1873), and other European countries. It appeared later in America, being found in the vicinity of New York in 1906 on *Ribes nigrum*, *R. rubrum*, and *R. aureum* as well as on *R. Grossularia* and *R. irriguum*, the plants of which had been imported from England (Stewart, 1906).

The sporidia from diseased leaves of *Ribes* infect the young shoots of certain varieties of *Pinus* growing in the vicinity, especially *Pinus Strobus* and *P. Cembra*, and to some extent also *P. Lambertiana* and *P. monticola*. At the end of July or the beginning of August, as a result of the infection, pycnidia are found in yellowish patches some 2–3 mm. in size. These patches can be seen through the cortex and from them spermatia are exuded in drops of sap which have a sweet taste. In the years following such an infection the cæoma stage of the fungus—*Peridermium Strobi*—breaks out on the bark in the form of transparent, colourless vesicles or blisters. After a few days the membrane covering the blister ruptures and the bright yellow masses of spores are freed.

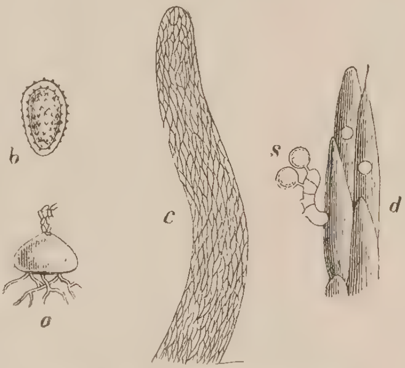

FIG. 162.—Currant Felt Rust. *a*, uredo-pustule; *b*, uredospore; *c*, teleuto-sorus; *d*, germinated portion of teleuto-sorus. (After E. Rostrup.)

The cæomaspores are able to infect the leaves of varieties of *Ribes* if such are in the vicinity, and after a few weeks give rise to the currant rust. After the cæoma blisters have discharged their contents, the affected parts of the bark die, as a rule, and acquire a corroded appearance. The mycelium hibernates within the stem and in the following year produces new lesions on the affected parts. After being subject to severe attacks of the disease for several years the twigs usually die, sometimes also young trees. In older plantations of pine the fungus works havoc.

The fungus has also another form of development: trees (13–26 years old) of white, or Weymouth, pine, in the neighbourhood of which no bushes of *Ribes* are growing, are found to have cæoma blisters on the 4–11-year-old parts of the stem and twigs, but not on the younger, 1–4-year-old, terminal parts of the twigs. From this it may be concluded that it is here a case of a 3–4 years' incubation period, and that a direct infection by cæomaspores from the pines themselves to the last annual shoots must be accepted. It has further been observed that when white pines are raised from imported seed the plants remain healthy for the first three or four years, and then, in the fourth or fifth spring, the blister rust appears on the lower, older branches. In each succeeding year the disease returns until after 6–8 years, if not sooner, most of the trees are dead. Probably in such cases the seed came from diseased trees, although they might appear healthy, and the disease was present in the seed and required an incubation period of several years before becoming ripe.

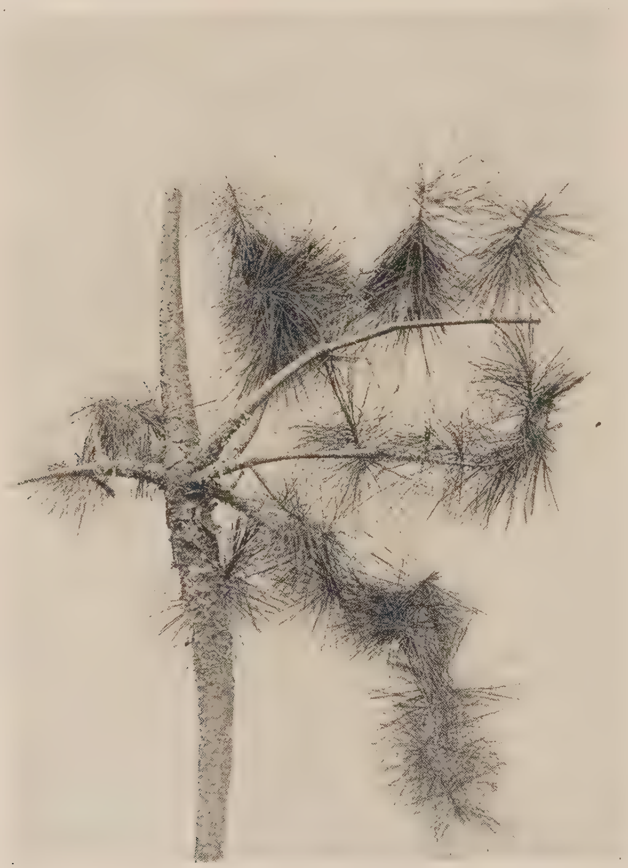

Fig. 163.—Currant Felt Rust. Æcidial stage (*Peridermium Strobi*) on *Pinus Strobus.*

Finally, it has been observed that when new bushes of *Ribes nigrum* are planted in a garden in which for years no stage of the fungus has been observed, nor in the vicinity, they may remain healthy for 3–5 years and then be suddenly attacked on the under side of the leaves by the fungus in the 4–6 years, at first only slightly but more severely in each succeeding year. It may be mentioned that in such cases the outbreak occurs relatively late in the year, in August.

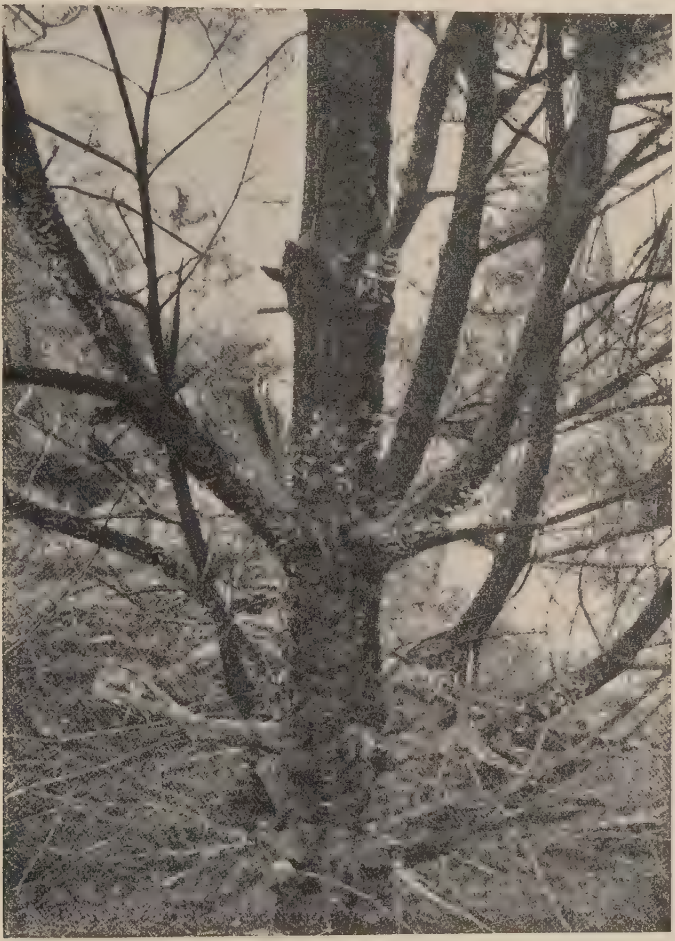

Fig. 164.—Currant Felt Rust. Æcidial stage (*Peridermium Strobi*) on *Pinus Strobus* in an American forest. (After Hampshire, 1919.)

In view of these observations, which have been made chiefly in Sweden, the opinion was expressed at the end of last century that the fungus can live from year to year without change of host, and can spread from place to place both in the form on the currant bush and in that on the white pine (Eriksson, 1896, 1922).

Considerable opposition has, however, been raised to this view (Klebahn, 1897, 1904; v. Tubeuf, 1901). After very exhaustive experiments in America and after the discovery of the rust in districts where no pines could be found, as in Canada (Quebec) in the years 1916–1921 (Dickson, 1921), and finally after comparison of the development of the rust with closely related forms such as *Peridermium Pini* (Wallr.) Kleb. in Europe (Haack, 1914; Klebahn, 1918) and *P. Harknesii* Moore in America (Meinecke, 1916, 1920), it appears that the fungus has both a heterœcious and homœcious method of development (Stewart and Rankin, 1912; Howitt and McCurbin, 1914; Stewart, 1917). It is regrettable that this view was not accepted at an earlier date, for then the white pine forests in America would perhaps have been saved from this destructive pest.

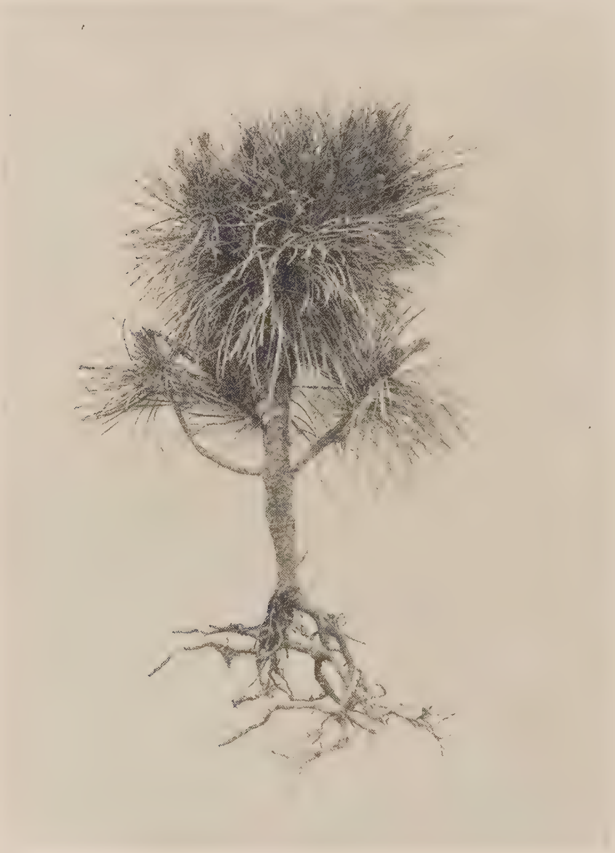

Fig. 165.—Currant Felt Rust. Æcidial stage (*Peridermium Strobi*) on a seven-year-old plant of *Pinus Strobus* (Stockholm). (Orig.)

This new view is supported by observations made on the period of incubation of the fungus in its attack on *Pinus*. Inoculation experiments in which the fungus was transmitted from *Ribes* to *Pinus* have shown that the period of incubation varies according to the age of the plant and the method of the experiment. Thus on young pines when the inoculation was made on the stem-parts the period was usually 5 years —1st year, inoculation ; 2nd year, dormant period ; 3rd and 4th years, swelling of stem ; 5th year, formation of cæoma (æcia) (McCurbin, 1917). If, however, the inoculation was made on the needles—the trees being older (15–30 years) or younger—development was more rapid, and in the following year cæoma blisters appeared on the one to two-year-old needles. After another year, swellings (canker) on the stem-parts were observed (Snell and Rathbun-Gravatt, 1925).

The injury caused to the *Ribes* plants is relatively unimportant when the disease is produced without change of host, as it then occurs late in the year, the middle or end of August, that is, at a time when the fruit has developed on the bushes and even begun to ripen. If, however, the bushes are in proximity to infected pines the injury is greater. When the blister rust breaks out on the pines in April the disease appears on the currant bushes in May and causes a considerable reduction in the amount of fruit. The fungus causes great damage to the white pine in countries where this tree is of considerable economic importance and leads even to its extermination in some cases, as, for example, in the forests of the eastern parts of the United States, where this pine is "one of America's most valuable timber trees" (Spaulding, 1912). It was estimated in 1919 that the value of these white pine forests in the United States and Canada was 1,121,750,000 dollars. In the autumn of 1921 the fungus, in spite of all control measures, had spread to the west (British Columbia, Puget Sound region in Washington State) and had severely attacked *Pinus monticola* and *P. Lambertiana*, the most important conifers in that region. It may be noted that about three-fourths of the estimated supplies of five-needle pines in the United States exist in the forests of the West.

For more than 100 years the white pine has been grown as an ornamental tree in European gardens and parks, and it was free of the disease until 1880. The fungus was spread in Europe through seeds and seedlings of *Pinus Strobus* imported from Russia, or from Switzerland (Upper Engadine), where it is found on *Pinus Cembra*, and develops its further stages on many species of *Ribes*, preferably *Ribes nigrum*, *R. petræum*, and *R. alpinum* (Schellenburg, 1923).

At the beginning of the present century the fungus reached North America, where the white pine is indigenous and was healthy, through seedlings imported from Europe (Germany and France). In 1909, about 2½ million and later more than 4 million seedlings were planted in various parts of the eastern States (Martin, Gravatt, and Posey, 1921) ; for three or four years they appeared healthy, in the fifth and sixth years disease broke out. Since then the disease has spread "more rapidly than was estimated previously " (Detwiler, 1920) and seriously threatens the large white pine forests.

Control Measures :

In Europe, where the white pine is only grown as an ornamental tree, it has been largely abandoned as the disease spoils its appearance ; it is also hoped that in the absence of the pine it will be easier to keep currant bushes healthy.

In North America, on the other hand, where the main hosts for the cæoma stage of the fungus—*Pinus Strobus*, *P. monticola*, and *P. Lambertiana*—are the chief commercial conifers, another method of control has been adopted, namely, the extermination of all species of *Ribes*, whether wild or cultivated, in the pine-growing districts. For this campaign, large numbers of people, including school children, are employed ; for example, in Ontario (Canada) no less than 9,342 children were engaged in 1917 ; in 1919 up to 252,114 acres were cleared of 4,574,293 currant bushes. It remains to be seen whether in this way the unattacked forests can be saved.

In 1922 the opinion was expressed that in view of the unsatisfactory position it would be best to obtain, as soon as possible, a variety of pine which is more resistant to this fungus and other diseases and introduce it into the forests (Eriksson, 1922). Recently this question has been under consideration in America, and it is believed that it may be possible to replace the disappearing five-leaved pines in the American forests by the Rumelian white pine *Pinus Peucc* Griseb. (v. Tubeuf, 1926).

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Pine Rust

Peridermium Pini (Willd.) Kleb.

This fungus attacks the twigs and branches of *Pinus silvestris* and is found on trees of all ages. It appears in early summer, often starting in the upper portion of the head of the tree. The attack generally begins on one side of the twig or branch and gradually spreads until the twig is girdled. On young twigs where the bark is thin the fungus gives rise to yellowish-white, vesicular growths very similar to those of the blister rust on white pine. On older twigs with a thicker bark the fungus often produces scattered spore-masses which lie in the crevices of the bark. After a period of some years the part of the branch above the seat of the disease dies, and in a few years afterwards the death of the tree follows.

This fungus is distinguished biologically from other forms of *Peridermium* by not having a change of host. Recent investigations have shown that, as was long supposed, this form of fungus can pass from one pine to another without the intervention of any intermediate host. After a period of incubation of 2–4 years new peridermium pustules are produced.

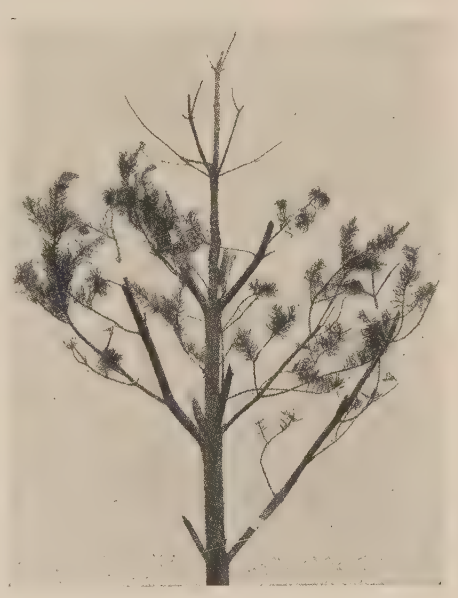

Fig. 166.—Pine Rust. Apex of pine tree killed by fungus. (After Haack.)

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Cronartium flaccidum (Alb. & Schw.) Wint. The uredo and teleuto generations of this fungus appear in the course of the summer and autumn as a dark brown felt on the under surface of the leaves of a number of species of the genera *Pæonia*, *Cynanchum*, *Asclepias*, *Nemesia*, *Sceptrum*, *Impatiens*, *Tropæolum*, *Verbena*, *Loasa*, etc., in all, on plants belonging to seven different families. The æcidial stage (*Peridermium Cornui* Kleb.) is developed on the stem of *Pinus silvestris*. The disease is known in most European countries and pæonies attacked by it suffer severely in some cases (Cornu, 1886 ; Klebahn, 1890–1904 ; E. Fischer, 1896–1904).

Cronartium gentianeum Thum. has been found on the leaves of *Gentiana asclepiadea* in Austria and Bulgaria. **C. quercinum** (Cooke) Miyabe develops uredo-pustules and teleuto-sori on the under surface of the leaves of species of *Quercus* and the æcidial blisters on a number of species of *Pinus*.

Coleosporium

Æcidia are found as long white threads on the needles of spruce. Æcidiospores united in long chains, uredo and teleuto generations in yellow to orange-red waxy masses on the under surface of the leaves of plants belonging to various families.

Pine Needle Rust

Coleosporium Senecionis Schum.

Peridermium Pini α acicola Wallr.

This fungus attacks the newly developed annual shoots of pine trees of different ages. When the disease is severe almost all the needles of the shoot are covered on both sides with long, yellowish-white æcidia ; very severe attacks bring about the death of young seedlings.

The uredo-sori, which develop on other hosts, are naked and pulverulent, at first yellow to orange-red, finally almost white with the uredospores in chains and sometimes with tube-like paraphyses. The teleuto-sori are flat, waxy, yellowish-red. The teleutospores, originally one-celled, soon divide into four superimposed cells, from each of which a sporidium is formed at the top of a long sterigma.

As most of the forms of *Coleosporium* are hardly to be distinguished morphologically, it is necessary to refer them to the host plant on which they live ; the European forms may be arranged in three groups : Group A (*Coleosporium Compositarum* Lév.) : (1) **Coleosporium Senecionis** (Pers.) Lév. on species of *Senecio* (*Peridermium oblongisporum* Kleb.) ; (2) **C. Tussilaginis** (Pers.) Lév. on *Tussilago farfara* (*P. Plowrightii* Kleb.) ; (3) **C. Petasitidis** (DC.) de By. on species of *Petasites*

(*P. Boudieri* Fisch) ; (4) **C. Inulæ** (Kunze) Fuck. on species of *Inula* (*P. Klebahni* Fisch.) ; (5) **C. Cacaliae** (DC.) Wagn. on species of *Cacalia* and *Adenostyles* (*P. Magnusianum* Fisch.) ; (6) **C. Sonchi** (Pers.) Lév. on species of *Sonchus* (*P. Fischeri* Kleb.). Group B (*Coleosporium Rhinanthacearum* Lév.) : (1) **Coleosporium Melampyri** (Reb.) Kleb. on species of *Melampyrum* (*Peridermium Soraueri* Kleb.) ; (2) **C. Euphrasiæ** (Schum.) Wint. on species of *Euphrasia* and *Alectorolophus* (*Peridermium Stahlii* Kleb.). Group C (*Coleosporium Campanulæ* (Pers.) Lév.) (*Peridermium Rostrupii* Kleb.) : (1) **Coleosporium Campanulæ-rapunculoidis** Kleb. on *Campanula rapunculoides*, *C. glomerata*, etc. ; (2) **C. Campanulæ-Trachelii** Kleb. on *C. Trachelium ;* (3) **C. campanulæ-rotundifoliæ** Kleb. on *C. rotundifolia*.

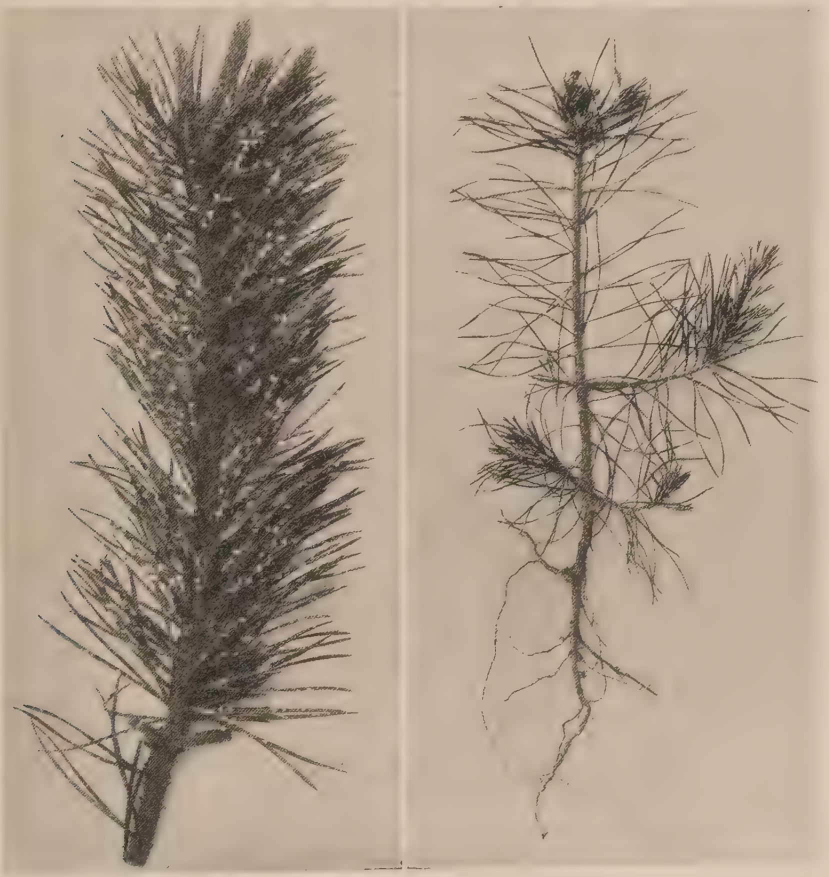

FIG. 167.—Pine Needle Rust. Æcidial stage (*Peridermium Pini a acicola*) on the shoot of an older tree and on a young seedling. (Orig.)

It must be mentioned, however, that the uredo and teleuto generations also occur where it has not been possible to find æcidial host plants, at least not any bearing æcidia.

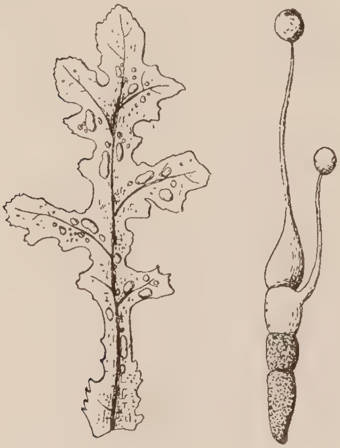

FIG. 168.—Pine Needle Rust. Teleuto-stage (*Coleosporium Senecionis*) on *Senecio vulgaris*. Diseased leaf and germinating teleutospore. (After T. Ferraris.)

Closely related to the above is **Ochrospora Sorbi** which develops æcidia (*Æcidium leucospermum* DC.) on abnormally long-stalked leaves of *Anemone nemorosa* and the subsequent stages on species of *Sorbus*, *Pyrus*, and *Spiræa*.

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Melampsora

Æcidiospores in chains with intercalary cells. Uredospores intermingled with clavate, thickened paraphyses. Teleutospores one-celled, cylindro-prismatic, placed side by side and forming a continuous brown to black crust covered by the epidermis of the host plant.

Pine-Poplar Rust

Melampsora pinitorqua (Braun) Rostr.

The æcidial stage of this fungus, formerly named *Cæoma pinitorquum* A. Br., attacks the woody portion of the annual shoots of *Pinus silvestris* and *P. montana*. At the end of May or the beginning of June, pycnidia appear on the shoots in the form of small, deep-yellow warts upon orange-yellow streaks. Soon afterwards the cæoma, in which the spores are arranged in rows, break through the bark in long, yellowish patches. This outbreak can easily be observed from the middle of June to the beginning of July on pines of all ages, but especially on those from 2–12 years old. Above the lesions—there may be several on each shoot —the shoot, if girdled by the disease, soon withers and remains in this state during the summer, hanging down as though injured by frost.

Resin, in varying amounts, is excreted from the edges of the wounds and gradually fills the cavities.

One-year-old seedlings, which are only rarely attacked, die almost without exception and two-year-old trees also suffer severely ; on this account the disease is very dangerous in nurseries. Older plants are not as a rule killed at once, the lower portions of the tree continuing to live and produce shoots to replace those that die. Complete recovery, even after repeated attacks of disease, sometimes occurs and may lead to an exceptionally good lot of trees being produced.

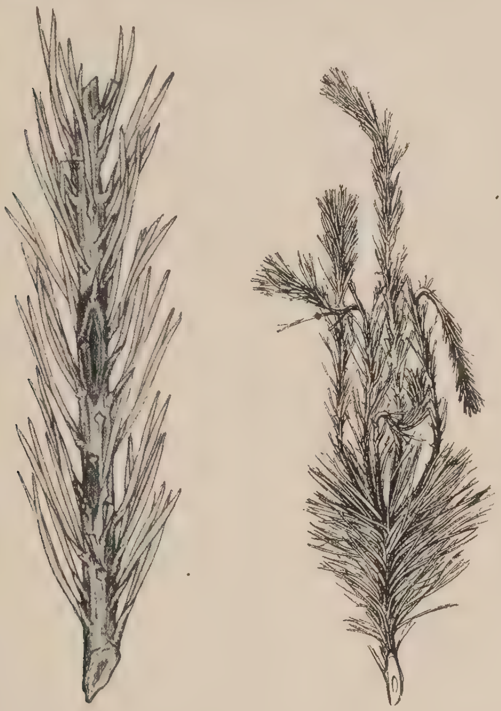

FIG. 169.—Pine-Poplar Rust.

a, twig of pine with lesions ; *b*, apical shoot injured by fungus. (*a*, after N. Sylvén ; *b*, after E. Rostrup.)

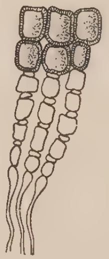

FIG. 170.—Pine-Poplar Rust. Three chains of spores. (After E. Rostrup.)

The cæoma-spores are able to germinate immediately after ripening and the germ-tube which is formed requires another host for its development. It enters the young leaves of *Populus tremula* and *P. alba* and after a few days gives rise to uredo- and teleuto-pustules. In the uredo-sori, which are small and yellow and all of them on the under surface of the leaf, swollen paraphyses are mingled with the spherical, or ellipsoid, echinulate spores. It is by means of these uredospores that the fungus spreads on the young poplar trees.

The teleuto-sori begin to appear in August on the under surface of the leaf, which soon acquires a blackened appearance. The teleutospores over-winter on the withered poplar leaves which fall to the ground in late autumn; they germinate in the following spring and can then infect any young pine trees in the vicinity. The ability of the teleutospores to spread the disease appears, however, to be very limited, and it has been observed in Sweden, in a nursery of eleven-year-old pine trees, that a few rows of the trees can protect the rest from infection by the spores.

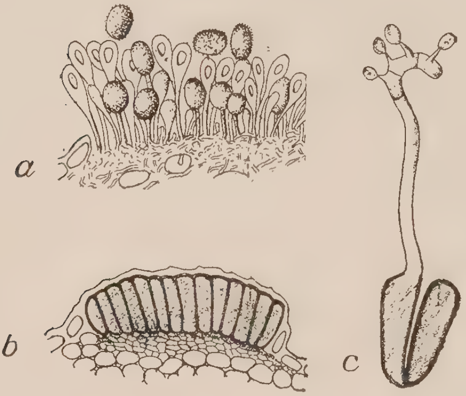

Fig. 171.—Pine-Poplar Rust. Uredo- and teleuto-stages. *a*, uredospore; *b*, teleutospores; *c*, teleutospore, germinated. (After G. Delacroix and A. Maublanc.)

The most dangerous form of the fungus—the cæoma generation—was first observed in 1860 in Hanover; it has since appeared with serious results in most European countries, especially in Northern Europe. When the change of host plant was shown (in 1884) it was easy to understand why the disease was especially prevalent in forests which had been planted after fires, as in such cases young poplar plants are common.

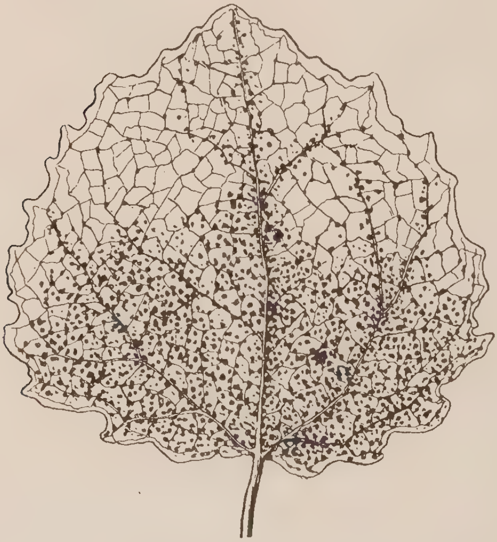
Fig. 172.—Pine-Poplar Rust. Teleuto stage on *Populus tremula*. (After N. Sylvén.)

Control Measures:

(1) Poplars should not be allowed to grow near to pines. (2) Large and exposed blocks of pine trees should be avoided. (3) Mixed plantations of spruce and pine are preferable.

The three following forms of fungi, all of which have the uredo and teleuto generations on the poplar, are closely related to the one just described: **Melampsora Laricis** R. Hart. with cæoma on the needles of *Pinus Larix;* **M. Rostrupii** Wagn. with cæoma on *Mercurialis perennis;* and

M. Magnusiana Wagn. with cæoma on species of *Chelidonium* and *Corydalis;* further also **M. Allii-populina** Kleb. and **M. Larici-populina** Kleb. both with the uredo and teleuto generations on *Populus balsamifera*, *P. canadensis*, etc., the first of these two last forms with cæoma on species of *Allium* and the second with cæoma on *Larix decidua*.

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Willow Rust

Melampsora salicina Lév.

The various species of *Melampsora*, which are found on different varieties of willow, will be dealt with under the above collective name. The uredo generation appears in the middle of summer, often to such an extent that the majority of the leaves acquire a yellow appearance in 2–3 weeks' time. The leaves on the lowest parts of the shoots are first affected, the others being attacked in acropetal order. On some willows the outbreak is on the under surface of the leaf and in others on the upper. The teleuto generation appears much later—about the time the leaves begin to fall—and the spores ripen after the leaves are on the ground. Where the attack is very severe, whole shoots of the willow may wither by the middle of summer.

There are a large number of specialised forms of *Melampsora* on willows which can only be distinguished biologically; most of them are heterœcious with the cæoma generation on plants belonging to widely differing families. The following heterœcious forms may be noted: (*a*) with cæoma on species of *Larix:* **Melampsora Larici-Capræarum** Kleb. on *Salix Caprœa*, **M. Laricis-pentandræ** Kleb. on *S. pentandra* and *S. fragilis*, and **M. Laricis-epitea** Kleb. with several specialized forms on numerous species of *Salix;* (*b*) with cæoma on species of *Allium:* **M. Salicis-albæ** Kleb. on *S. alba* and **M. Allii-fragilis** Kleb. on *S. fragilis*, etc.; (*c*) with cæoma on *Galanthus nivalis*, **M. Galanthi-fragilis** Kleb. on *S. fragilis*, etc.; (*d*) with cæoma on species of *Ribes:* **M. Ribesii-viminalis** Kleb. on *S. viminalis* and **M. Ribesii-purpurea** Kleb. on numerous species of *Salix;* (*e*) with cæoma on species of *Euonymus:* **M. Euonymi-Capræarum** Kleb. on *S. Caprœa*, etc.; (*f*) with cæoma on numerous *Orchideæ*: **M. repentis** Plowr. on *S. repens*; (*g*) with cæoma on *Saxifraga aizoides*: **M. reticulata** Blytt. on *S. reticulata*; (*h*) with cæoma on *Saxifraga oppositifolia*: **M. alpina** Juel on *S. herbacea*. Many of the forms of *Melampsora* found on the willow occur in places where the appropriate cæoma host is absent and cannot even be discovered in the neighbourhood.

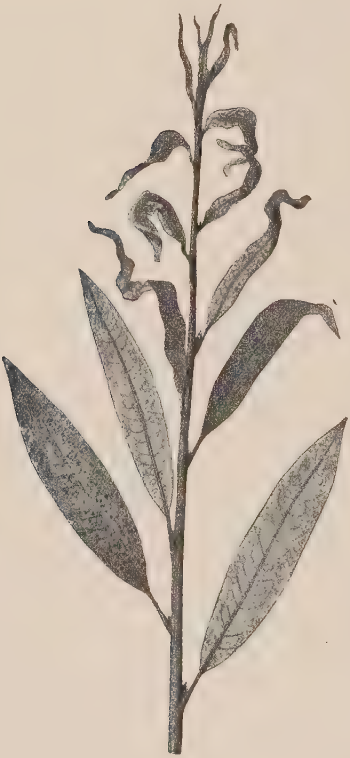
Fig. 173.—Willow Rust. (After E. Rostrup.)

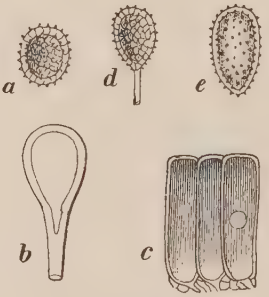

Fig. 174.—Willow Rust.

a, uredospore; *b*, paraphysis; *c*, teleutospores on *Salix purpurea*; *d*, uredospore on *Salix purpurea*; *e*, uredospore on *Salix enspidata*. (After E. Rostrup.)

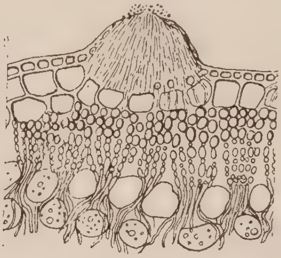
Fig. 175.—Willow Rust. Æcidial stage on *Pinus silvestris*. Cross-section of a pustule. Above a spermagonium, below æcidia. (After R. Hartig.)

There are also some autœcious forms such as **Melampsora Amygdalinæ** on various species of *Salix*, **M. Hypericorum** (DC.) Schröt. on numerous species of *Hypericum*, and **M. Helioscopiæ** (Pers.) Wint. on species of *Euphorbia*, etc.

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Melampsoridium betulinum (Pers.) Kleb. produces, in the middle of summer, small crowded uredo-sori on the under surface of the leaves of *Betula odorata* and *B. verrucosa*. In autumn the dark brown teleutosori appear—also on the under surface of the leaf—and the teleutospores are able to produce a cæoma stage on the leaves of *Larix europœa* (Rhoads, Hedgcock, Bethel, and Hartley, 1918).

Canker or Witches' Broom of Silver Fir

Melampsorella Caryophyllearum Schröt.

Æcidium elatinum Alb. & Schw.

The æcidial stage of this disease, known formerly as *Æcidium elatinum* Alb. & Schw., is the cause of injury to *Abies pectinata* as it leads to the formation of large canker-like swellings on the stem and twigs of young and old trees. These cankers enlarge from year to year and often give rise to deep fissures in the bark. If a bud is situated on a canker, then, in the following spring, it develops into a witches' broom which grows vertically upwards and continues in this manner for at least five or six years, producing what looks like a small tree. The leaves on the witches' broom are of a light, yellowish-green colour and æcidia are found in two

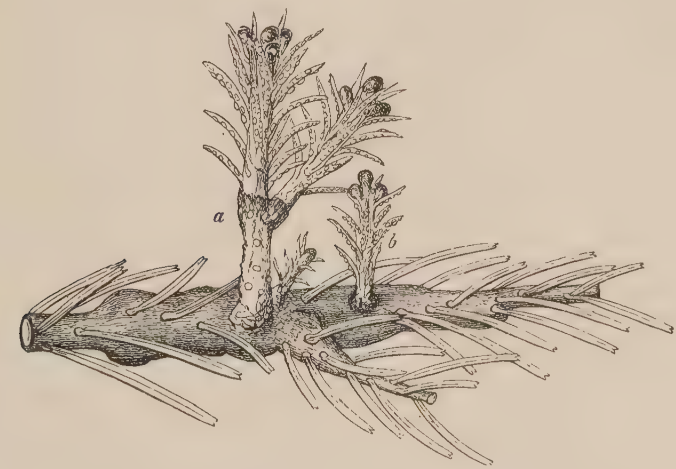

FIG. 176.—Silver Fir Canker. Twig with two-year-old witches' broom (*a*). At (*b*) a dormant bud forced into growth. (After R. Hartig.)

irregular rows on the under surface. In late autumn, the leaves of the witches' broom fall and the shoot stands bare in the winter. The fungus continues to live, however, in the cankers, and these increase in size each year. Such a witches' broom may go on growing for more than twenty years and the cankers themselves for more than sixty years; the latter are sometimes found without witches' brooms being present on the tree. The twigs attacked by the canker often become so brittle that they break off in a strong wind or under the weight of snow. It is easy to understand that the economic value of trees attacked by this fungus is much reduced.

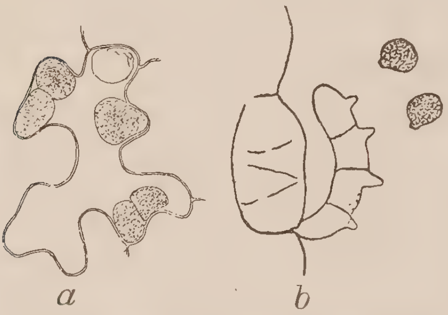

Fig. 177.—Silver Fir Canker. Teleuto stage (*Melampsorella Caryophyllearum*) on *Stellaria nemorum*. *a*, ungerminated spore; *b*, germinated spore and sporidia. (After E. Fischer.)

The æcidiospores ripen in June or July and infect the leaves and shoots of numerous species of the genera *Alsine*, *Arenaria*, *Cerastium*, *Stellaria*, etc., on which they produce uredo-sori in from 10–14 days. The mycelium lives on in these host plants if they are perennial and develops teleutospores in the following May. The teleutospores are unicellular, developed within the epidermal cells and provided with a thin, colourless membrane; they germinate with promycelia and sporidia.

As the vegetative system of this fungus, in the æcidial generation as well as in the uredo and teleuto generations, lives perennially in the host plant it is easy to understand that the two generations are often found widely separated from one another in nature.

The disease is found wherever fir trees are grown, and it sometimes attacks other varieties such as *Abies Nordmanniana*, *A. cephalonica*, and *A. Pinsapo* in Europe, *A. Pichta* in Siberia, and *A. balsamea* in North America.

To control the disease it is recommended to cut off the witches' brooms, particularly those on young trees.

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Pucciniastrum Abieti-Chamenerii Kleb., also called *P. Epilobii* Otth., develops its uredo and teleuto generations on the leaves of *Epilobium angustifolium*, *E. latifolium*, and *E. Dodonei*. The uredo-pustules, either singly or in groups, appear on the under surface of the leaf as yellow spots. The upper surface of the leaf where diseased has a yellowish-red appearance. The teleuto-sori, which are also found on the under surface of the leaf, are mainly in groups, and they finally cause the whole of the lower surface to become brown to black. The teleutospores, which germinate in the spring, give rise to an æcidial stage on the under side of the needles of *Abies pectinata* and *A. balsamea*, the æcidia being arranged in two rows. Needles which are severely attacked by the fungus fall prematurely (Klebahn, 1899, 1903 ; v. Tubeuf, 1902 ; Fraser, 1912).

The closely related species **Pucciniastrum Epilobii** Otth. occurs on other species of *Epilobium* such as *Epilobium alpinum*, *E. hirsutum*, *E. tetragonum*.

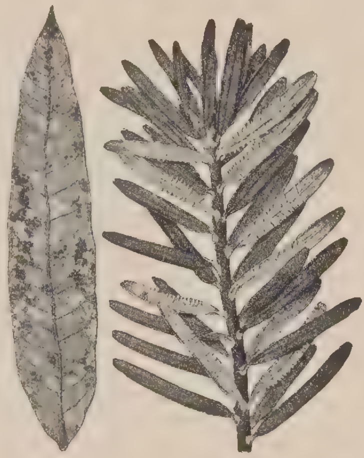

Fig. 178.—*Pucciniastrum Abieti-Chamenerii*. Left, a leaf of *Chamenerion augustifolium* with light (uredo) and dark (teleuto) spots. Right, a twig of fir with æcidia. (After E. Rostrup.)

Thekopsora areolata (Fr.) Magn., also named *Pucciniastrum Padi* (K. & S.) Dietr., appears very frequently on the leaves of *Prunus Padus*, *P. serotina*, and *P. virginiana*. The uredo-sori are found on the under surface of the leaf as small, pale warts or spots which are pink-red below and dark brown above and are sharply limited by the small veins of the leaf. The teleuto-sori, which come later, are in the form of dark brown crusts, mainly on the upper surface of the leaf. The teleutospores are divided by thin longitudinal walls. These spores on germination can produce an æcidial stage on the cones of *Picea excelsa* and *P. obovata* (*Æcidium strobilinum* Rees). The æcidium gives rise to collections of brown warts on the inner side of the scales. The æcidiospores germinate in the following May and can then infect the young leaves of *Prunus* (v. Tubeuf, 1902 ; Fischer, 1902, 1905).

Thekopsora Vacciniorum Karst., also named *Pucciniastrum Vacciniorum*, produces yellow uredo-sori and brown, crust-like teleuto-sori on the under surface of the leaves of *Vaccinum Myrtillus*, *V. uliginosum*, *V. Oxycoccus*, etc. In America the forms which are found on *Vaccinium canadense* and *Gaylussacia resinosa* have been transmitted to *Tsuga canadensis* (Fraser, 1913, 1914).

Calyptospora Gœppertiana Kuhn. develops the æcidial stage, *Æcidium columnare* Alb. & Schw., as yellow spots on the under side of the needles of *Abies alba*, *A. balsamea*, and *A. Fraseri*, and the teleuto stage on abnormally lengthened and thickened shoots of *Vaccinium Vitis Idæa*, etc. The lower leaves of the whortleberry fall prematurely.

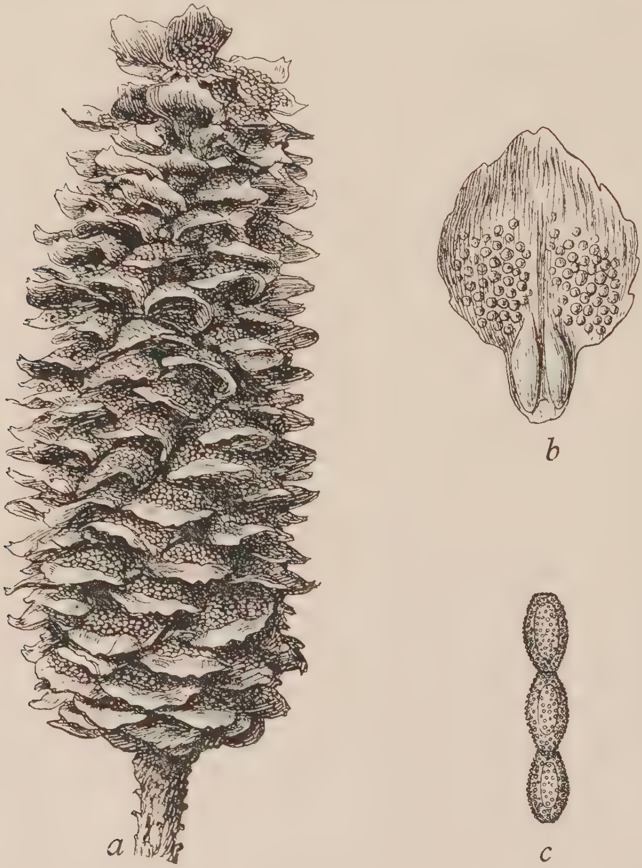

FIG. 179.—*Thekopsora areolata.*

a, spruce cone with æcidia on inner side of scales; *b*, a single scale; *c*, three members of a chain of spores. (After E. Rostrup.)

The mycelium seems to be able to hibernate in the stem of the teleuto host.

Melampsora Lini Pers. produces at first yellow and later rust-red to black sori on the leaves and stem of flax (*Linum usitatissimum*). In severe attacks the stems of the plants become brittle and are then not suitable for the preparation of flax. The fungus on common flax is a single specialised form of the collective species and has been also named *Melampsora liniperda* (Körn.) Palm. Other species of *Linum*, such as *L. alpinum*, *L. catharticum*, and *L. tenuifolium*, have each their specialised forms of the fungus (Palm, 1910; Fromme, 1912; Buchheim, 1915; Hart, 1926).

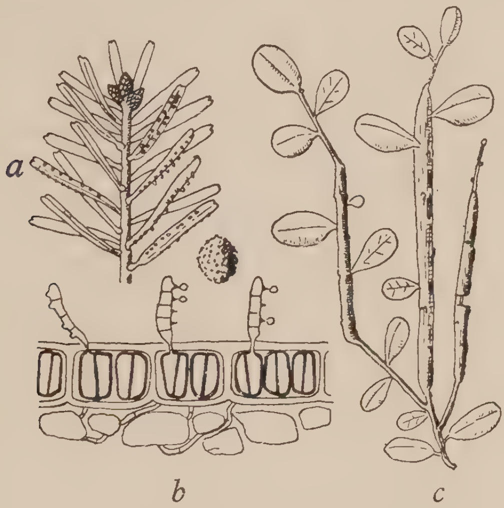

FIG. 180.—*Calyptospora Gœppertiana.*

a, shoot of fir with æcidia and single æcidiospore; *b*, germinating teleutospores; *c*, deformed shoot of whortleberry. (After F. W. Neger.)

CHAPTER VII

HYMENOMYCETINEÆ

THE fruiting bodies (sporophores) vary considerably in size and structure, being in some cases a felt of mycelium and in others a firm mass of tissue. The mycelium puts out side branches (basidia) from the tips of which basidiospores are abstricted.

Agaricaceæ

The spore-forming hymenium is composed of lamellæ or gills on the under surface of the cap or pileus. The cap is usually fleshy and does not persist. There is a stalk or stipe which is usually central, rarely eccentric or lateral. The mycelium as a rule lasts for several years, spreads in the ground in all directions, and from it young branches send up rapidly-growing sporophores to the surface.

Honey Fungus

Armillaria mellea (Vahl.) Quél.

The sporophores of this fungus—sometimes called honey agaric or shoestring fungus—appear in groups in late autumn, especially after the first night frosts. The sporophores are found on the old trunks of foliage trees and conifers, on dead branches lying on the ground, and sometimes they appear on the ground itself. The sporophores form at first domed, honey-yellow caps which later enlarge and become yellowish-brown in colour ; the caps are covered with brown fibrous scales. At about two-thirds of the distance between the cap and the ground a white membranous annulus is formed on the stalk. The flesh and the lamellæ are white, as are also the spores.

In the wood and bark of diseased stems and roots there are found brown or black strands of mycelium which may run regularly through the tissues or may branch and anastomose. These strands, which are luminous at night, are called rhizomorphs and are formed of medullary tissue with an outer cortex. From the young peripheral cortex there are developed delicate threads of mycelium which penetrate into the wood of the stem. Formerly, a distinction was made between (1) *Rhizomorpha subterranea*, which spreads in the soil and often develops a sporophore at the apex, and (2) *Rhizomorpha subcorticalis*, which produces white, branched sheets of mycelium between the cortex and the wood.

The wood parenchyma, especially in the case of conifers, is seriously damaged by the mycelium, the cell walls being gradually disintegrated. In the end the tree becomes withered and dead, the diseased wood breaking up into cubical pieces. The boundary between healthy and diseased wood is often marked by a black line.

This disease, which may be regarded as the most dangerous one in the forests of Mid- and Northern Europe, was first described scientifically in 1790 under the name of *Agaricus melleus* L. (Vahl). The rhizomorph stage has been known since 1797 (Roth). The connection between the two stages—the rhizomorphs and the sporophores—as well as the general life-history of the fungus was shown in 1873 (R. Hartig). The mycelium arising from the rapidly germinating spores spreads like a network in the soil and can infect the roots of the tree under favourable conditions of moisture. Gradually the mycelium assumes the firm, leathery form of the rhizomorph, and in this form the fungus lives saprophytically on decaying tree-stumps or on posts standing in the ground. The disease attacks the stem from the outside and spreads inwards, thus differing from the attack of polyporus, which starts in the centre.

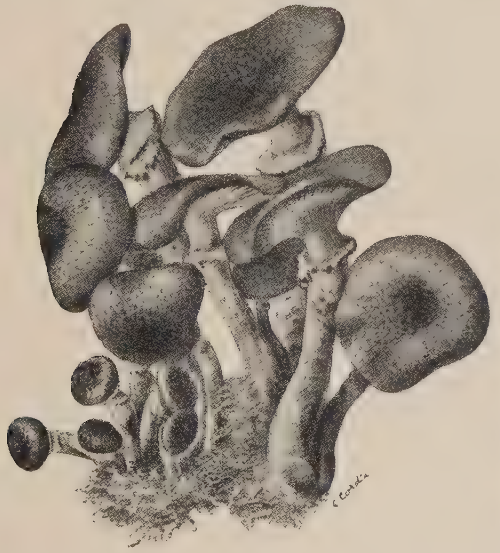

FIG. 181.—Honey Fungus. Group of sporophores in various stages of development. (After E. Rostrup.)

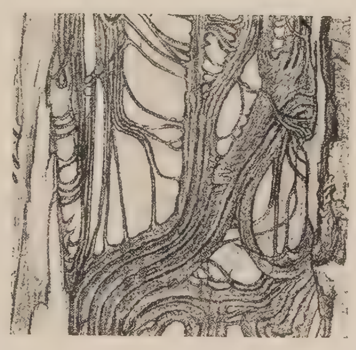

FIG. 182.—Honey Fungus. Rhizomorph stage from stem of alder. (After E. Rostrup.)

Most trees are susceptible to this fungus but in varying degrees; many conifers, and particularly those in young forests and on poor soil, are attacked by it. In Denmark it was found that *Picea excelsa*, *Pinus silvestris*, *P. montana*, and *P. Strobus* were most susceptible, whereas *Picea alba*, *Pinus Cembra*, *Larix europœa*, and *Juniperus communis* were only rarely attacked (Rostrup). In general, *Picea excelsa* suffered most, particularly when grown on soil from which beech trees had been cleared. The rhizomorphs left behind in the soil probably flourish on the material which remains from the beeches and then spread to the roots of the young conifers and infect them.

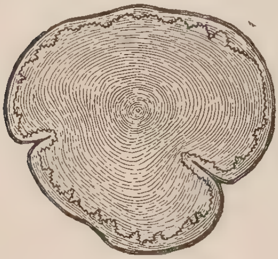

FIG. 183.—Honey Fungus. Cross-section of stem of beech killed by fungus. The black line shows the extent of the penetration of the mycelium into the wood. (After E. Rostrup.)

Observations made in Denmark have shown that *Fagus*, *Carpinus*, *Populus canadensis*, *Salix alba*, and *Sorbus Aucuparia* are readily attacked; *Quercus*, *Betula*, and *Fraxinus* more rarely; whilst *Tilia*, *Corylus*, *Sambucus*, *Æsculus*, *Cytisus*, *Pyrus Malus*, *Prunus Cerasus*. *Populus tremula*, *P. pyramidalis* only exceptionally become infected.

In Western and Southern Europe, also outside Europe, this fungus is known; in England on *Pyrus Malus*, *Ribes Grossularia*, and *Rubus fruticosus;* in Holland on *Prunus Cerasus;* in France on *Castanea*, *Juglans*, and *Olea;* in the Pyrenees on *Betula* and *Castanea;* in Italy on *Morus;* in North Africa on *Ficus Carica;* in Uganda on *Theobroma*, *Coffea*, and *Thea;* in North America on *Quercus*, *Pyrus Malus*, and *Prunus Cerasus;* in Java and Sumatra on *Thea*, etc.

Control Measures:

(1) Trees that are attacked by the rhizomorphs but which can still be put to use should be felled at once, as they depreciate each year. (2) If the fungus has recently appeared and to a limited extent, the spread of the rhizomorphs may be checked by digging a deep, narrow trench ($1\frac{1}{2}$ feet) round the infected tree or group of trees. (3) In plantations where the disease appears, the sporophores should be collected and destroyed so as to prevent the spores spreading the infection. (4) If susceptible trees are to be planted in cleared beech woods all old stumps, etc. (for which the rhizomorphs have a preference), should be carefully removed.

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Collybia velutipes Curt., sometimes called "the winter fungus" because the sporophores do not decay even during the autumn frosts, but revive and grow again in winter when a thaw sets in. The fungus has yellow to reddish-brown sporophores which are often grouped together, and they are found on the stems of fruit trees, elms, limes, oaks, willows, poplars, etc. Rhizomorph strands occur between the cortex and the wood of the stem. The fungus gives rise to a brown decay of the stem and it can also live saprophytically.

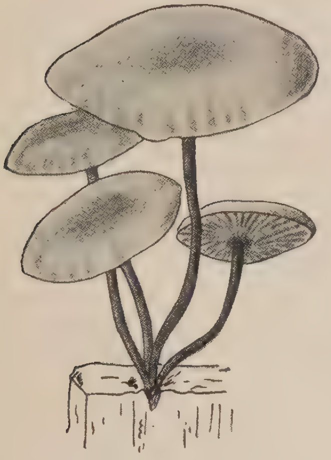

FIG. 184.—*Collybia velutipes.* (After F. W. Neger.)

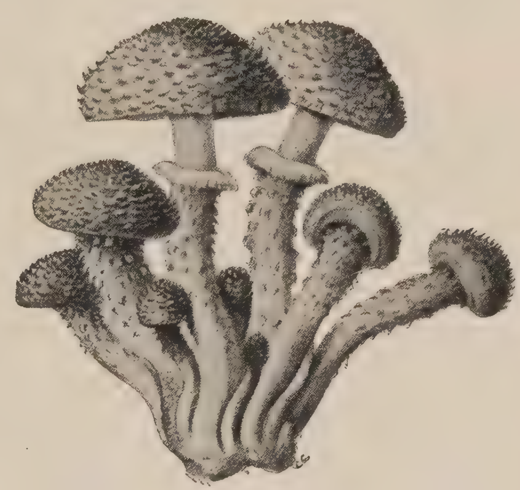

FIG. 185.—*Pholiota squarrosa.* Sporophores in various stages of development. (After E. Rostrup.)

Pleurotus ostreatus Jacq. This fungus appears on wounds and places where branches have broken off. A number of broad-leaved trees are attacked, especially willow and poplar, sometimes lime, birch, etc. The sporophores are blue-black to brown-grey on the top, the stalk is either absent or misshapen, short, thick, with stiff hairs. The caps are grouped together and can withstand frost, the lamellæ and spores are white. The wood gradually crumbles into thin, white, paper-like sheets, and the stem ultimately becomes hollow.

Pholiota squarrosa (Mill.) Karst. forms large, crowded collections of sporophores at the base or on the higher parts of the stems of beech, ash, poplar, lime, elm, apple, cherry, etc. The sporophores are stiff, straw-yellow, the cap domed and closely covered with reflexed scales, as is also the stem. The lamellæ (gills) are olive-green, later dark brown. The wood is destroyed by white rot.

Pholiota adiposa Fr. lives on the stems of beech and cherry and also on conifers. The cap is yellow and slimy with reflexed, detachable scales, the stem sulphur-yellow, the gills at first yellow and later brown. In the case of the white pine the wood splits into lamellæ along the annual rings, giving the appearance of having been traversed by boring insects (Neger, 1919). **Ph. aurivella** Batsch. found on beech and apple. The scales of the cap as well as those of the stalk are pressed together; the stalk is sticky. **Ph. destruens** Brond. with white scales on the cap and stalk; it is found on the stem of poplar.

"Fairy Rings" on Grass.—These occur on meadows and lawns and are characterized by a ring of strongly-growing grasses of a deep-green colour. This ring is surrounded by an outer one in which the grass has died more or less completely. In the outer ring, toadstools of various kinds arise, sometimes growing so closely together that they press against one another. The fairy rings increase in diameter from year to year, the part in which the toadstools are found spreading outwards. The mycelium grows centrifugally in the soil in all directions, whilst the inner, older portions gradually die. The toadstools, which soon die down, serve as a manure for the grass, and this accounts for the luxuriant growth which is seen in the inner rings. In the central part of the ring the grass grows badly owing to lack of food.

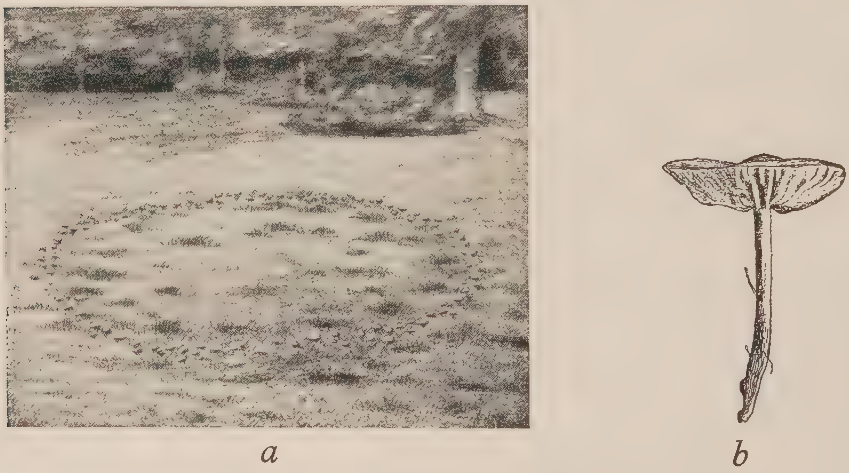

Fig. 186.—Fairy Rings on a Meadow.
a, fairy ring caused by *Marasmius Oreades*; *b*, the fungus. (After E. Rostrup.)

The following species are concerned in the formation of fairy rings: *Agaricus campestris*, *Marasmius Oreades*, *Clitocybe gigantea*, etc. (Rostrup, 1902; Bayliss, 1911).

Polyporaceæ

The spore-bearing hymenium lies on the inner side of narrow, closely-packed tubes or on the folds of labyrinth-like passages. The pileus varies in form, being either flat, bracket-like, or hoof-shaped, and grows horizontally from the trunk of the tree, or else from the ground, in very irregular shapes. The pileus is sometimes soft and soon decays, or else leathery, corky, or woody and not infrequently persists for several years.

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Polyporus vaporarius Pers.

This fungus produces a very thin, white, expanded sporophore which adheres firmly to its substratum. The sporophore has a tree-like appearance and is composed of isolated hyphæ along with thicker strands. It is found on the trunks of living conifers and on the wood of such trees in cellars, glasshouses, etc. The free, upper surface of the hymenium is covered with numerous irregular pores from the walls of which colourless basidiospores are abstricted. The fungus spreads as a white, snow-like mass over the layer of dead needles found in pine and fir forests, attacks the roots through wounds and grows on from these points. It is distinguished from the true dry rot (*Merulius lacrimans*), with which it is sometimes confused, by having a permanent, white mycelial membrane (Wehmer, 1925).

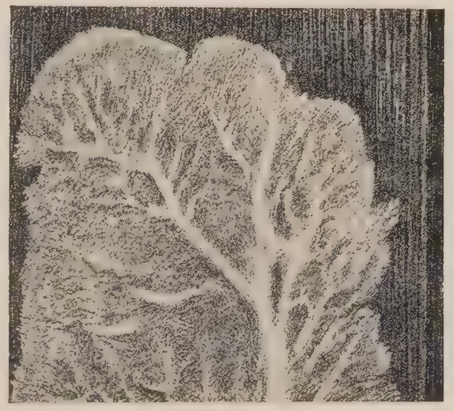

Fig. 187.—*Polyporus vaporarius*. Mycelium. (After E. Rostrup.)

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White Rot of Foliage Trees

Polyporus squamosus Huds.

The sporophores are kidney-shaped, often of very large size; they have a short stalk and stand out horizontally. At first they are fleshy, later leathery or woody and with brown scales on the upper surface. The fungus attacks living trees of nut, horse chestnut, apple, pear, elm, oak, willow, poplar, beech, lime, maple, etc. The fungus is found more frequently on trees in gardens and avenues than in forests. The diseased wood undergoes white rot.

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Polyporus sulphureus Bull.

The sporophores, which are sulphur-yellow to orange in colour, are in the form of closely overlapping, shelf-like growths, one above another. They arise from places where branches have broken off or from other wounds on the stem. At first, the sporophores are soft and watery, later they become leathery; the upper surface is reddish-yellow and the flesh white; on the sulphur-yellow under surface are numerous pores. In addition to the colourless basidiospores formed on the inside of the pore tubes, grape-like groups of conidia or chlamydospores arranged in chains arise from the mycelium within the stem. After a few weeks the sporophores become brittle and crumble away. The wood develops red rot and splits along the medullary rays or annual rings into cracks, which appear as white streaks in longitudinal and cross-sections.

This fungus attacks the oak preferably and finally produces a hollow trunk. Not infrequently it also attacks apple, pear, cherry, hazel nut, acacia, chestnut, *Gleditschia*, *Prunus serotina*, and *Salix alba*.

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Polyporus betulinus Bull. develops its sporophores on young, dead birch trees as well as on older living ones. The sporophores are hoof-shaped, sometimes with a short stalk; the upper surface is grey-brown, the lower one white; the flesh is white and the spores colourless. In the fresh state, the layer of tubes can be easily separated from the pileus; when dry the pileus is fragile and brittle. The fungus gives rise to a red rot in the wood (this rotten wood is rubbed down to a powder and used for polishing in the Swiss watch industry). To limit the spread of the fungus in birch woods it is recommended that badly affected trees should be removed or at least that the sporophores should be sawn off and burnt (Rostrup, 1883; Mayr, 1884; Vanin, 1922).

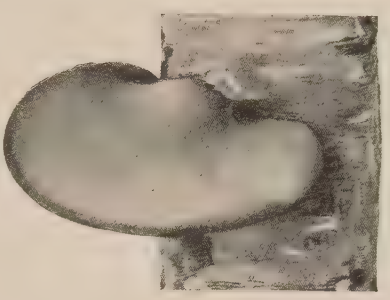

FIG. 188.—*Polyporus betulinus.* (After E. Rostrup.)

Polyporus connatus Fr. gives rise to white, thick, irregular sporophores which are often massed together in large numbers. The fungus attacks poplars, especially *Populus canadensis*, sometimes also horse chestnut, maple, elder, and birch. The flesh of the sporophore is white and the spores colourless. The fungus causes decay of the heart wood and makes the trunk hollow.

Polyporus radiciperda Hart.

This fungus, known also as *Trametes radiciperda* (Hart.) Rostr., is the most dangerous parasite found in conifer forests. The attack of the fungus begins on the under side of the roots, the fungus avoiding the light. The sporophores, which vary in size from a pea to a hand's breadth, are plate-like in shape and attached to the roots. The upper surface of the sporophore is brown, somewhat lighter in colour at the margin, and the inside is white. Each year a new layer of pores is added to the older one. The fungus reproduces itself either in autumn by means of spores which have only a short period of germination, or by mycelial hyphæ which grow at all times of the year and spread the disease through the soil from the roots of one tree to another. In the case of *Picea excelsa* a layer of individual or coalescing crusts forms on the under side of the thin roots and, later, on that of the thicker roots. In *Pinus silvestris* and many other trees the sporophores are found at the base of the trunk, often in rings round the tree. From the root, the fungus ascends the stem, sometimes reaching a height of from 15–20 feet.

The diseased wood has at first a greyish-violet colour, later becoming reddish-brown with scattered, elongated, black spots surrounded by a white margin. In the lower parts of the trunk there is a considerable secretion of resin and, in consequence, the fungus does not in some cases ascend further, but grows on in the branches of the root and kills them. The wood breaks up into spongy strands which have an unpleasant smell, resembling that of urine. The attack of the fungus causes trunks of large trees to become hollow.

Generally speaking, the older trees are more resistant to the fungus; those of 6–10 years of age generally die in the first year of the attack, whilst a 20-year-old tree may live for three or more years. A violent storm will bring down affected trees, and in 1894 it was estimated that in Denmark 300,000 trees, which were still alive but rotten, were blown down.

The fungus attacks numerous conifers—spruce, pine, fir, thuja, and juniper; the white, or Weymouth, pine and the Douglas fir appear to be particularly susceptible. Certain foliage trees are also attacked by the fungus, *e.g.* beech about 5–20 years old, especially when the trees are growing on ground that still carries stumps of conifers. When a beech tree is attacked it often puts out supplementary roots above the diseased roots so as to make good the loss. Old beeches are seldom attacked and more rarely still cherry, oak, mountain ash, ash, white thorn, pear, etc.

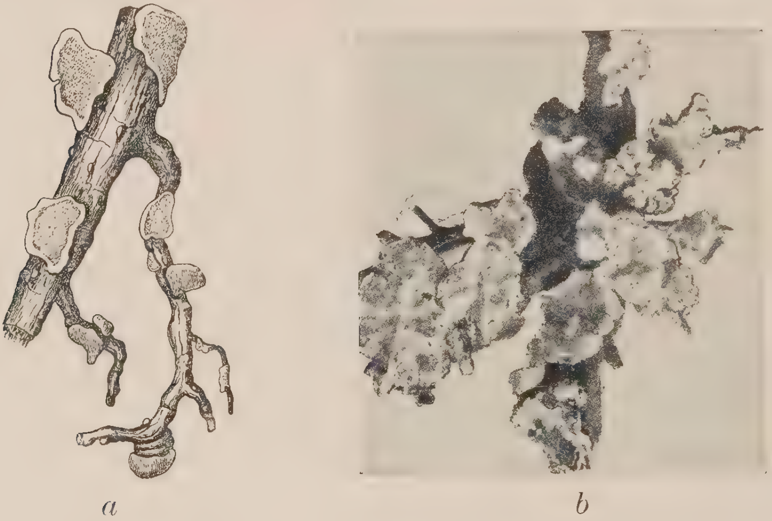

Fig. 189.—*Polyporus radiciperda.* Sporophores on the under side of the roots: *a*, in early stage; *b*, in later stage. (*a*, after R. Hartig; *b*, after F. W. Neger.)

Control Measures:

(1) Avoid planting too closely. (2) Plant conifers and foliage trees in mixed stands. (3) In slight attacks, dig a narrow trench round the

infected trees so as to check the spread of the mycelium and fill this trench with loose earth. (4) Cut off all sporophores that appear above the ground and burn them. (5) Fell the trees in badly diseased stands, the value of the timber decreases each year.

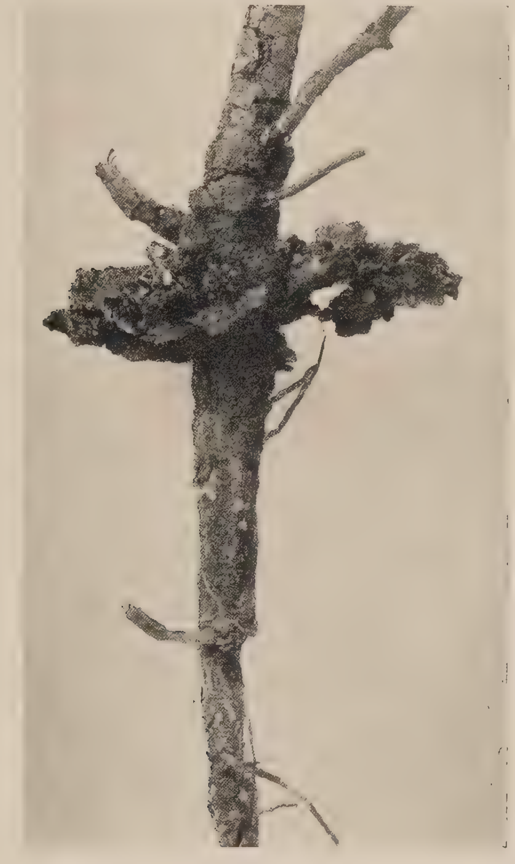

FIG. 190.—*Polyporus radiciperda.* Young plant of *Pinus silvestris* killed by fungus. (Orig.)

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Polyporus fomentarius L.

This is one of the most widely-spread of the fungi which attack wood. The sporophores, of which a number may be present on one trunk, are bracket- or hoof-shaped and sometimes of a very large size. The upper surface of the sporophore is of an ash-grey colour and concentrically toothed, the under surface white, later light brown, the flesh brown, the spores colourless.

The fungus is parasitic on beech, poplar, and elm, more rarely on other trees such as birch, alder, and horse chestnut; it produces white rot of the wood. The mycelium causes the wood to split into thick, white, radial pieces, destroys the cambium in places, and often produces longitudinal furrows on the trunk from which the sporophores can arise. The boundary between diseased and healthy wood in the trunk is marked by black lines. A soft, but tough, mass of hyphæ is formed inside the bark, and formerly this was treated with water, wood ash, and saltpetre, and after drying used as tinder, etc.

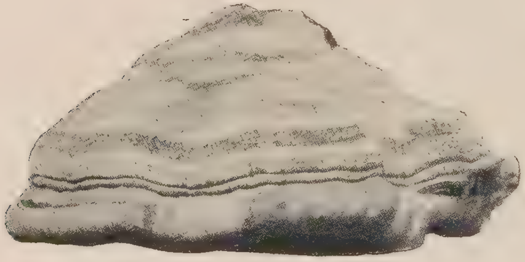
Fig. 191.—*Polyporus fomentarius.* (After F. W. Neger.)

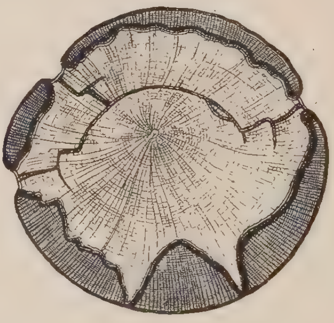
Fig. 192.—*Polyporus fomentarius.* Section of beech stem killed by fungus. The dark peripheral layer is healthy; in a few places the fungus has reached the outside and caused cracks to appear. (After E. Rostrup.)

It is almost impossible to save a beech tree that is badly diseased, consequently it should be felled so as to use as much of it as possible and prevent the spread of the fungus. If the tree has to be retained the sporophores should be sawn off and burnt.

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Polyporus igniarius L. is found on stone-fruit trees. The sporophores are hoof-shaped and very hard, the upper surface greyish-black, domed in shape and often cracked; the lower surface is moderately arched. After some years the upper surface exhibits numerous concentric grooves. The interior of the sporophore is also very hard, the flesh brown, the spores colourless. Sometimes the fungus appears on oak, poplar, and willow, more rarely on hazel, birch, and *Hippophaë*. To prevent the spread of the fungus to other trees, the sporophores must be cut off as soon as they appear (Münch, 1915; Rudan, 1917). **P. dryadeus** Fr. is found at the base of the trunk and on the roots of the oak. The sporophores are hoof-shaped, at first soft and watery, later corky; they give rise to yellow and white streaks in the heart-wood. **P. Ribis** Schum. produces leathery, bracket-like sporophores at the base of old gooseberry and black currant bushes. As a result of the attack, the crop is reduced and the berries are small.

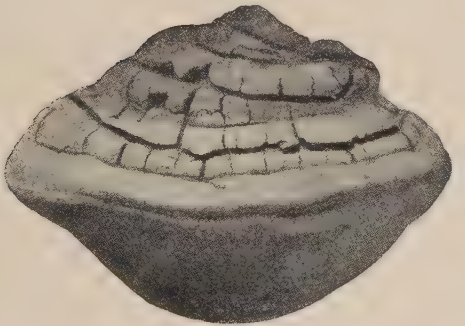

FIG. 193.—*Polyporus igniarius*. (After F. W. Neger.)

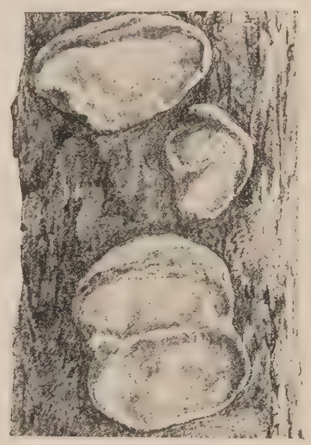

FIG. 194.—*Polyporus fulvus* var. *Oleæ*. (After E. Prillieux.)

Polyporus fulvus Fr. var. *Oleæ* R. Hart. This fungus attacks the olive tree (*Olea europæa*), especially the older stems. Round or elongated patches appear at places on the stem where there are depressions in the bark. The spores of the fungus are carried by wind to cracks or wounds and there germinate. On cutting through such a lesion, the peripheral tissue of the stem is seen to be coloured brown into the cambium, where a considerable amount of mycelium is found. The mycelium produces sporophores on the surface of the stem and these are irregularly circular, at first, sericated and brownish-yellow, later grey. The disease is widely spread in all the olive-growing countries of Southern Europe.

At first, the mycelium lives saprophytically upon the wood which it dissolves, and later the cambium and the cork are attacked. The starch disappears from the woody tissues and its place is taken by a brown, gummy substance. The cell walls are destroyed and the whole of the tissue is split up into irregular portions, being finally changed into a lint-like mass.

The following measures are recommended to control the disease :

(1) Paint all open wounds on the stem with a solution of sulphate of iron and then cover them with tar. (2) Cut out, down to the healthy wood, all diseased patches, as soon as they appear. (3) Cover the wounds where patches have been cut out with tree-banding grease or similar material. (4) Destroy the sporophores (R. Hartig, 1893 ; Prillieux, 1895).

Polyporus radiatus Sow. produces large numbers of small sporophores which are of a rust-brown colour on the outside and inside. The sporophores lie one above another like tiles on a roof and form long, aggregated crusts on the stem. The fungus is parasitic upon alder, sometimes also on beech, but rarely on birch, elm, hornbeam, maple, and hazel. **P. sistotremoides** Alb. & Schw. forms short, thick sporophores on pine, spruce, Douglas fir, and white pine. The sporophores are at first yellowish-brown and soft, later they become dark brown and corky ; they are arranged like roof tiles (imbricate) on the stem. The wood is changed to a reddish-brown colour with chalky-white sheets of mycelium and it finally becomes so rotten that it can be crumbled to a yellow powder between the fingers. **P. nigricans** Fr. attacks birch mainly. It does not produce a sporophore of regular shape but only irregular, fissured, black nodules without a pore layer. On the willow and poplar, normal sporophores are developed.

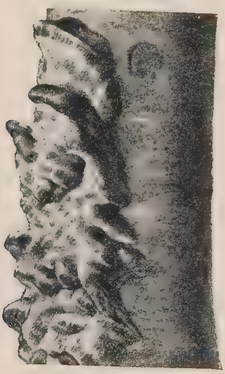

Fig. 195.—*Polyporus radiatus.* (After E. Rostrup.)

Polyporus Pini (Thore) Rostr.

This fungus, which is also named *Trametes Pini*, forms relatively small, corky to woody crusts, or brackets, of sporophores. The upper side of the sporophores is at first dark brown and later becomes almost black, with concentric furrows ; the lower side is a dirty yellow at first and finally brown.

As a rule, the fungus is only found in forests where the trees are at least fifty years old. Infection takes place at points where branches have broken off and there the sporophores develop. The decayed wood is rust-brown with white patches, but without the black streaks of mycelium found in the case of *P. radiciperda*.

The fungus attacks various conifers—pine, spruce, fir, and larch.

Owing to the decay of the wood the trees are often broken by wind. The disease is very common in the north of Germany and in Northern Europe, and it is also found in the south of Germany; in Prussia it is estimated that in the State forests damage to the extent of a million marks occurs annually.

Control Measures :

(1) To prevent infection every care should be taken to avoid any injury being caused to the green branches and to the bark of the stem. (2) Trees attacked by the fungus should be cut down, or at least the sporophores removed and the places covered with tree-banding grease so as to prevent the formation of new sporophores.

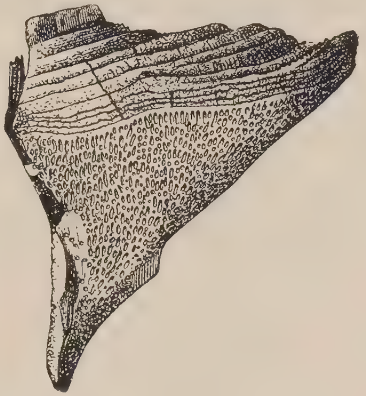

FIG. 196.—*Polyporus Pini.* (After R. Hartig.)

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MÖLLER, A. Der Kampf gegen den Kiefernbaumschwamm. Zeitschr. Forst- u. Jagdw., 1910, 129–146; Ib., 1914, 193–208.

Polyporus hispidus (Bull.) Fr. forms yellowish-brown, fleshy sporophores, which are bristly on the upper surface, on broad-leaved trees such as ash, elm, beech, mulberry, and some kinds of fruit trees. The spores are brown and a white rot is produced in the wood.

"Esca" Disease of Vine

Stereum necator Viala.

In the vine-growing countries of Southern Europe and in the whole of the Mediterranean area, certain vines have been observed, from earliest times, to suddenly become diseased. This often occurs in July or August and only a few vines in the vineyard may be attacked. The leaves, which until then had been green and turgid, turn to a yellow colour, acquire a mosaic appearance, droop, and wither. The grapes and the tendrils of the vines also dry up and die. The disease attacks older vines (20–25 years), more rarely younger ones (4–15 years). The suddenness of the attack is indicated by the name given to it in France —"Apoplexie."

The disease is due to the development inside the stem of a fungus belonging to the group Hymenomycetes—*Stereum necator* Viala, which is sometimes found living together with *Polyporus igniarius.*

Owing to the formation of tinder-like plugs of mycelium which block the vessels transporting the food material, the ascent of the sap is suddenly interrupted, and, in consequence, the leaves and fruit of a branch suddenly die or the whole vine may be killed. Only rarely, and after careful treatment, do the vines recover in the following year. The disease occurs most commonly on cold, damp soils.

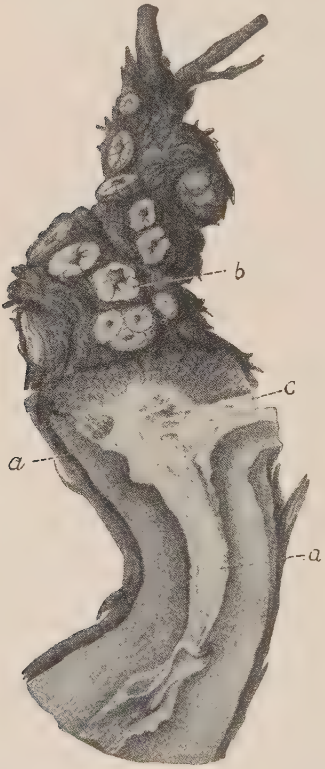

Fig. 197.—Esca Disease of Vine. Median longitudinal section showing black-bordered filling.

a–b, wounds caused by pruning; *c,* a pruning wound at point of entry of fungus. (After P. Viala.)

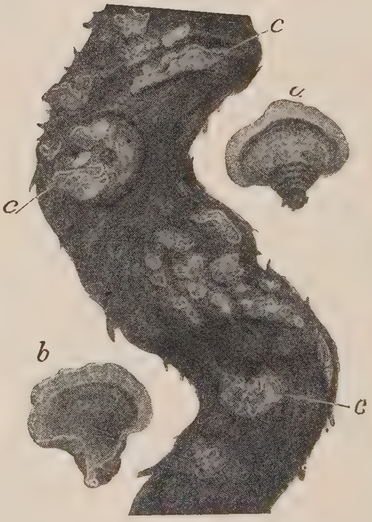

Fig. 198.—Esca Disease of Vine.

a, sporophore of *Stereum necator* seen from below; *b,* seen from above; *c,* part of stem with groups of sporophores. (After P. Viala.)

The fungus enters by way of a pruning-wound at a time (August) when the circulation of nutrient material is very active. The mycelium first penetrates the medullary tissue of the wound, staining it deeply, almost black, and spreads both laterally into the peripheral tissues and downward to the neck of the root. A ferment (oxidase) is secreted which impregnates the cells of the wood and kills them. The mycelium grows very slowly in the first 4–5 years and no outward signs of the disease are visible, but it finally fills the whole of the central cylinder of the stem with a tinder-like mass so that the foliage suddenly dies, which is usually an indication that the vine will be killed. Physiological abnormalities in the growth of the vine are contributory factors in the disease.

The fungus provides for its continued existence and dissemination in various ways. Only rarely, about one case in a thousand, old vines develop sporophores in October or the beginning of spring. These sporophores are small, round, or of irregular shape, brown and leathery with concentric zones; they are arranged in groups on the stem. Usually the sporophores do not bear basidiospores, being only annual, and, having regard to their rare and weak development, they probably play no important part in the economy of the fungus.

The reproduction of the fungus and the spread of the disease from plant to plant appears generally to be achieved by means of lamelliform sclerotia (" lames sclerotiques ") which are formed by the mycelium. When these sclerotia germinate they send out delicate, tube-like hyphæ which produce endoconidia and in this way bring about infection through wounds on the stem.

The disease has been known from earliest times in the vineyards on the Mediterranean, especially in Syria, Palestine, Asia Minor, Macedonia, Smyrna, Crete, Samos, Corfu, etc., under the name of " Iska " (Greek) or " Escha " (Italian), which means " tinder." It is now found in all vine-growing countries except perhaps South America, South Africa, and Australia. In Sardinia (Squinzano) about 32 per cent. of the vines perished from this cause in the years 1904–13. In France the spread of the disease and the damage which it causes may be placed on a level with that due to *Uncinula necator* (*Oidium Tuckeri*) and *Plasmopara viticola* (Viala, 1926).

For the prevention and control of the disease it has been found, in France, that spraying, after pruning, with a fungicide containing arsenic (20 kg. white arsenic, 15 kg. soda, and 100 litres water; this mixture to be further diluted 1 part to 15–20 parts of water before spraying) gives good results. Spraying must be carried out for two years, either consecutively or with an interval of a year. By such treatment the spores of the fungus on the wounds caused by pruning are destroyed, and the tinder-like aggregations of mycelium in the central cylinder are impregnated and disinfected.

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St. purpureum Pers. produces a silvering of the upper side of the leaves of a number of trees such as stone fruits, plane, chestnut, lilac, buckthorn, gooseberry, black currant, rose, etc., hence the name "Silver Leaf." The disease spreads from the infected root through the whole plant, but no mycelium has been found in the infected leaves. The silvering appears to be due to some secretion of the fungus which is transmitted from the seat of infection.* A badly diseased tree usually dies within 3–6 years. The sporophores occur one above the other in tiers (imbricated) on the trunk of the trees, sometimes densely crowded together; they may be villose or tomentose. The hymenium is naked and of a purple-red colour (Cotton, 1921, 1922; Brooks and Storey, 1923; Brooks and Moore, 1923; Ferdinandsen, 1923). **St. hirsutum** Fr. attacks the living wood of felled oaks and changes it into white zones or streaks which, in cross-section, appear as rows of white dots and in longitudinal section as white lines. The sporophores are in the form of crusts or are raised from the trunk, the upper surface yellowish-brown, roughly hirsute with yellow margins. **St. quercinum** Pott. found at the base of dead branches of oak as canker-like lesions from which small sporophores arise.

Fistulina hepatica Huds., Beef-steak Fungus, develops large, tongue- or kidney-shaped sporophores, the upper surface blood-red, later brown, the under side with finely-spined pores. The fungus lives on the trunks of older oak trees and stains the wood a reddish-brown colour at the points where the sporophores are attached; it is sometimes found on the trunk of the chestnut. **Thelephora laciniata** Pers., **Th. terrestris** Ehrh., **Th. fimbriata** Schw. give rise to large patches of semicircular sporophores of a soft, leathery nature on the soil. The sporophores are found in woods where the air and soil are very moist, and especially in seed-beds, where they may be so thickly congregated that the young plants are smothered (v. Tubeuf, 1905; Weir, 1921).

Aureobasidium Vitis Viala & Boy. produces small, dark spots on ripening grapes. These spots enlarge until at least one-third of the skin is affected. The mycelium ramifies through the flesh of the grape causing it to dry up. On the dried patches minute, bright yellow, cottony sporophores appear. The basidia develop six or more sterigmata, the basidiospores are elongated and cylindrical. The leaves gradually lose their green colour and turn red. This disease was first observed in France, 1882–85, in Italy and Istria, 1896–97; the form of the fungus which appears there is the variety *album* which has white sporophores, not yellow, and causes the whole vine to die (Viala and Boyer, 1891; Viala, 1893; Osborn and Samuel, 1922; Darnell-Smith, 1922).

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* The silver colour is also stated to be due to the separation of the epidermis of the leaf from the underlying tissues.

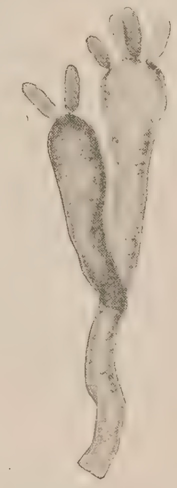

Fig. 199.—*Aureobasidium Vitis.* (After P. Viala and G. Boyez.)

Rhizoctonia Disease of Potato

Hypochnus Solani Prill. & Del.

Rhizoctonia Solani Kühn.

Potato plants attacked by this disease show, in summer, a thin, white or reddish-grey felt round the stems a few inches above the ground; the felt in some cases passes down below the surface. It is composed, mainly, of colourless, branched hyphæ which here and there give rise to bunches of basidia from which basidiospores are produced. The underground parts of the stems, the stolons and roots, are also attacked by the fungus. The tubers are more or less covered with brownish-black, hard crusts (sclerotia) which may be ½ cm. in diameter but are usually smaller. In this stage on the tubers, the fungus is named *Rhizoctonia Solani.* In damp soils or in those deficient in humus, the fungus is able, by means of the sclerotia, to over-winter or to pass through long periods of drought.

As a rule, the fungus does not cause any serious injury to the stem or to the tubers. The sclerotia do not penetrate into the tubers deeply and can be easily removed without leaving visible injury to the skin, although sometimes, owing to insect damage, the fungus enters to a greater depth. Latterly the opinion has been expressed that the disease may be responsible for considerable damage to the crop owing to the disorganisation of large portions of the stem, which adversely affects the interchange of food material between the parts of the plant above and below ground. It has also been observed that the embryonic tissues of the shoots and roots can be killed by the fungus.

In America, at least two different strains of the fungus have been distinguished. These strains differ biologically and also morphologically—dimensions of spores, hyphæ, sclerotial cells, etc. (Rosenbaum and Shapovalow, 1917; Matsumato, 1921).

A species of fungus belonging to the group of the Basidiomycetes was discovered in 1903 in Colorado living saprophytically upon the dead bark of various conifers. The spores on germination produce a mycelial felt similar to that of *Rhizoctonia*. The fungus has been named *Corticum vagum* B. & C. var. *Solani* Burt., and since its discovery it has been regarded in American

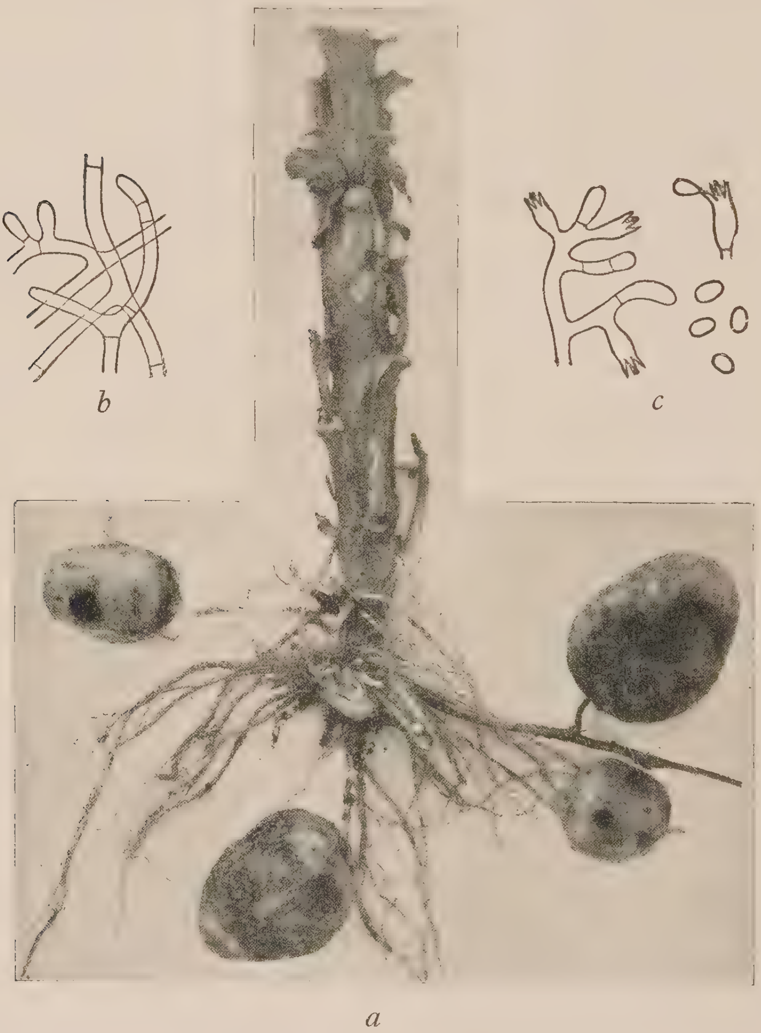

Fig. 200.—Rhizoctonia Disease of Potato.

a, white spore-producing felt (*Hypochnus Solani*) on potato stem, and black sclerotia-forming mycelium (*Rhizoctonia Solani*) on stolons, tubers, and roots; *b*, hyphæ from the white felt; *c*, basidia and basidiospores. (Orig.)

literature as the spore-bearing stage of the potato Rhizoctonia, even indeed as that of *Rhizoctoniæ* in general. As this conifer fungus has never been observed in Europe, the American view must be considered as very problematical, at least as regards European conditions.

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Typhula Disease of Cereals

Typhula graminum Karst.

This fungus has been observed on young plants of wheat, winter barley, and rye grass. The mycelium spreads throughout the little plant and causes its death ; sclerotia are found on the surface of the dead parts or in internal cavities. These sclerotia are hard and about the size of a seed of cabbage or clover, rarely larger, the upper surface white, becoming pale red ; inside they are dense and of a pure white. Finally, the sclerotia fall from the host plant, remain unchanged during the winter, and germinate in the following spring or summer ; they are thus the over-wintering stage of the fungus. On germination, the sclerotia put out one or more thread-like sporophores which may be simple or branched and are more or less expanded at the apex. Towards

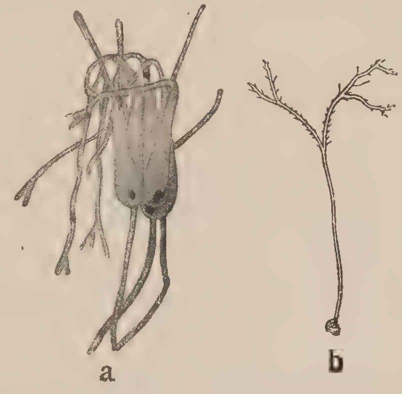

Fig. 201.—*Typhula graminum*. *a*, wilted wheat plant upon which are sclerotia ; *b*, sclerotium with branched sporophore. (Orig.)

the ends of the sporophores, basidiospores are abstricted which give rise to a new generation and cause fresh outbreaks of disease.

In the spring of 1878 the fungus appeared to a serious extent near to Stockholm on a variety of wheat (Mainstay) obtained from England; native varieties growing alongside were not attacked. Similar cases occurred in three other districts; in the worst case all the plants died.

In the spring of 1921 the fungus was general on winter barley in Brandenburg and Mecklenburg, the plants turning yellow and many of them dying. The application of nitrogenous manures (nitrate of soda and ammonium sulphate-nitrate) as well as hoeing and harrowing brought about the recovery of the plants and a good harvest was obtained (Zimmerman, 1922).

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Typhula Betæ Rostr. generally appears on the upper parts of sugar beets which are stored in clamps and intended for seed production. The fungus is also found on the stems and branches of growing sugar beet plants that are forming seed. The sclerotia are black and of the size and shape of a cabbage seed. This fungus has sometimes been very prevalent in Denmark on beets stored in clamps for the winter and is

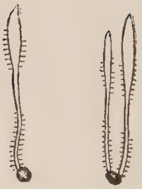

Fig. 202.—*Typhula Trifolii.* Sclerotia from which sporophores have developed.

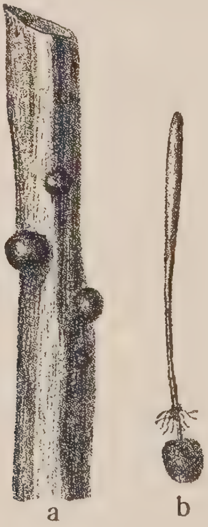

Fig. 203.—*Typhula gyrans.* *a*, leaf-stalk with sclerotia; *b*, sclerotium with sporophore.

also found, but more rarely, in other European countries such as Germany and Spain; it has been very destructive to the sugar beet crop in the Azores. The fungus causes the outer leaves to wither, then the leaves turn yellow and droop, forming a ring on the ground, even the heart leaves exhibit signs of yellowing, and finally the plant dies. When such a diseased beet is drawn from the ground it appears as though enclosed in a lump of soil (Rostrup, 1902; Molz, 1920).

Typhula Trifolii Rostr. attacks various species of clover. The tip of the host plant is permeated by a delicate mycelium and in a short time becomes black. The sclerotia are brown, then black, and of the size and appearance of clover seed (Rostrup, 1902).

T. gyrans Batsch. appears on plants of the cabbage family (cabbage, rape, turnip), both on the leaf, particularly the leaf-stalk, and on the root. The sclerotia are reddish-brown, becoming black and of the size of a cabbage seed. The disease has been observed in Denmark (Rostrup, 1866, 1902).

As a measure of protection, care should be taken that sclerotia are not present in the seed.

CHAPTER VIII

EXOBASIDIINEÆ

The basidia are subepidermal and break through the epidermis; they are unicellular, clavate, with four terminal sterigmata each of which bears a spore. These fungi give rise to noticeable galls on the parts of the plants which they attack.

Azalea Gall

Exobasidium Azaleæ March.

The leaves of *Azalea indica* become thickened, fleshy, and pale in colour in places; in some cases the leaf buds are deformed and are changed to thick, fleshy rosettes of leaves. On the diseased patches a chalky-white coating, resembling hoar frost, appears and is caused by the basidia with their basidiospores breaking through from below. Within these gall-like structures there is no differentiation of the leaf tissue into palisade- and spongy-parenchyma, all the cells are polyhedral, and there is an almost complete absence of chlorophyll granules. Each basidium regularly produces four spores.

The fungus was first described from Japan in 1896 as *Exobasidium japonicum* Shir, and it was probably introduced into Europe about 1906 on azalea plants sent from Japan. It was reported in various European countries—Belgium, Holland, Germany, Italy, etc.—about the same time, *i.e.* in the years 1906–09. In 1908 more than 300 azalea plants of Belgian origin were severely attacked in a nursery garden in Dresden.

Experiments carried out in Belgium have shown that some azaleas offer a certain resistance to the fungus, the best sorts being Raphael, Vervæneana, President Oswald de Kerchove, and Madame Petrick.

It is considered that a fly (*Aleurodes vaporarius*) plays an important part in the spread of the disease.

The removal and burning of all infected leaves as soon as possible is the only means of controlling the disease with success.

Similar galls are found on *Rhododendron ferrugineum* and *Rh. hirsutum* in Alpine countries and are produced by **Exobasidium Rhododendri** Cram.

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Exobasidium Vaccinii (Fuck.) Wor. appears on the leaves, stems, and flowers of some species of *Vaccinium*. On *Vaccinium Vitis Idæa* the fungus produces large blister-like swellings which are carmine-red on the upper part and covered below with a whitish-yellow bloom. Elongated, hypertrophied areas occur on the stem and on the flowers red and white variegated, abnormal growths. On *V. Myrtillus* the infected leaves are sometimes three or four times as large as normal and in some cases large portions of the wood are attacked. On *V. Oxycoccus* erect, pale red, swollen shoots arise from the creeping stem. Often the forms of the fungus appearing on different species of *Vaccinium* are regarded as separate species of the fungus (Juel, 1921 ; E. Fischer, 1916).

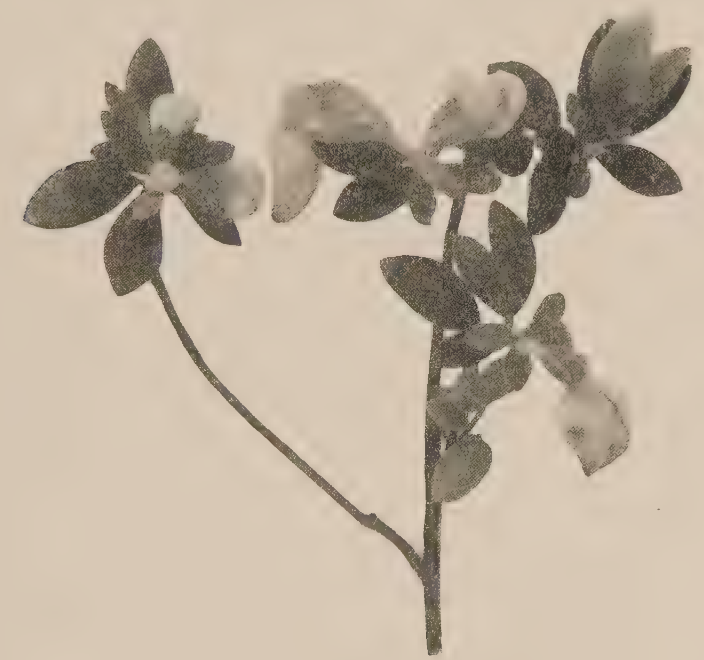

FIG. 204.—*Exobasidium Azaleæ.* (Orig.)

CHAPTER IX

ASCOMYCETES

THE chief spore-bearing structures are sporangia or asci derived from them.

Exoascaceæ

The asci, which arise from a subepidermal mycelium, appear as a layer over the surface of the parts of the plant attacked by the fungus. The spores are very resistant to cold, drought, and shortage of food. The fungus hibernates in twigs or buds. The fungi in this family give rise to various malformations—patches, swellings, hypertrophy, witches' brooms—on the leaves and twigs of a large number of different kinds of trees.

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Plum Pockets

Taphrina Pruni (Fuck.) Tul.

This fungus attacks the developing ovaries of *Prunus domestica, Pr. Padus,* and *Pr. virginium.* In consequence of the stimulation of the internal mycelium the young fructifications, after flowering, grow into long, crooked, one-sided, pocket-like structures. These are at first green and smooth but later become pale, compressed, wrinkled, and covered with a delicate greyish-white or ochre-yellow bloom. A fruit does not develop inside these structures; they remain empty and finally shrivel up and fall to the ground.

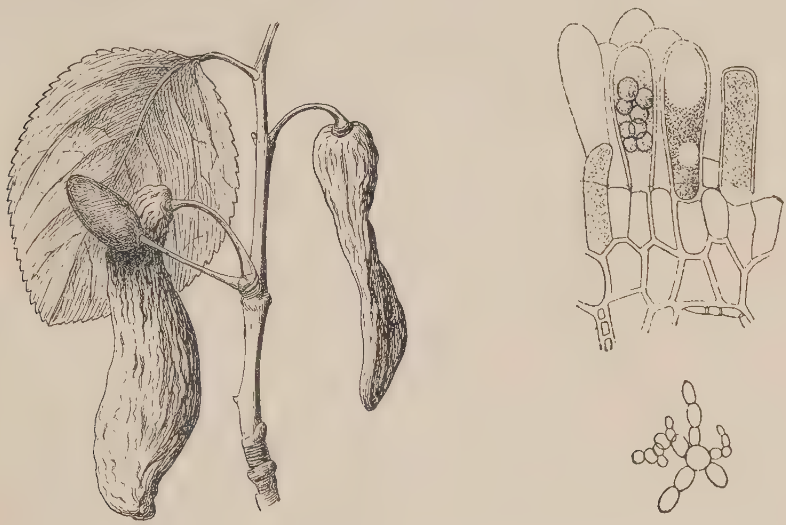

Fig. 205.—Plum Pockets. Left, a twig of plum with two pockets. Right, asci and spores, latter with yeast-like buds. (After E. Rostrup.)

On the surface of the plum pockets the asci are close together forming an uninterrupted hymenium. Protoplasm is found only in the upper part of the ascus, the basal part being cut off to form a stalk cell. In the protoplasm-filled cells, 6–8 ascospores are produced. The large numbers of spores freed from the asci give a flour-like appearance to the surface of the pocket. In water or in a nutrient solution containing sugar, the spores bud like yeast cells.

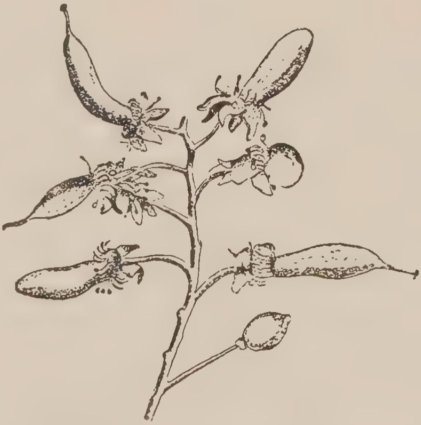

Fig. 206.—Plum Pockets on Bird Cherry. (After P. Sorauer.)

The tendency for the same tree to produce plum pockets year after year can be explained by the hibernation of the vegetative system of the fungus in the twigs.

In *Prunus Padus* the fructifications begin to change before flowering, and in consequence the calyx and stamens, together with the upper part of the petiole, are included in the plum pocket.

This disease seems to have been known to a number of older writers such as A. Cäsalpin (1519–1603) and R. J. Camerarius (about 1694). It was considered to be due largely to unfavourable, cold, wet weather at the flowering period. About the middle of the last century the disease was more closely investigated and described (Fuckel, 1861 ; de Bary, 1864). At the present time the disease appears to have spread to all the countries where these varieties of plums are grown.

Similarly deformed fruits, due to **Taphrina Rostrupiana** (Sad.) Gies., occur on *Prunus spinosa* and *P. insititia.*

Control Measures :

(1) Collect and burn all affected fruit. (2) Cut out—down to the older wood—all diseased branches. (3) Get rid of green fly (aphides) on the trees, as these insects favour the establishment of the spores of the fungus.

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Taphrina aurea (Pers.) Fr. attacks the tips of the twigs and the leaves of a number of species of poplar. Observations made in a nursery of poplars near to Stockholm in 1883 and 1884 showed that in the first year, at the end of July, some of the tips of the twigs of *Populus pyramidalis*, *P. canadensis*, *P. fastigiala*, etc., were swollen and bore bright yellow streaks. The leaves were golden-yellow and bladder-like, usually with the lower surface concave and covered with a golden-yellow hymenium, the upper surface convex and green. The undeveloped leaf structures at the tips of the twigs were entirely yellow and curved upwards. In the second year, the swellings and yellow streaks were absent from the tips of the twigs, but small blisters were found on a number of leaves. The infected leaves were distributed fairly regularly over the whole of the tree but the patches were less noticeable. On the diseased twigs the older, lower leaves were most affected ; the terminal leaves were healthy.

In this second year of the disease the species of poplar most affected by the disease were *Populus pyramidalis*, *P. canadensis*, and *P. Lindleyana*. Less affected were *P. nigra*, *P. fastigata*, *P. angulata*, *P. Eugenii*, *P. hybrida berolinensis*, and *P. monilifera*. Not affected at all were *P. balsamifera macrophylla*, *P. Simoni*, *P. alba*, *P. rotundifolia*, *P. tremuloides pendula*, and *P. laurifolia*.

Taphrina insititiæ (Sad.) Joh. attacks the leaves and twigs of *Prunus domestica* and *Pr. insititia*. The asci arise on the under surface of the leaf and give to it the appearance of being covered with a greyish-white bloom, the upper surface is puckered. The fungus gives rise to large witches' brooms, sometimes in great quantity, as in Sweden (Johanson, 1885) and Denmark (Rostrup, 1902). The fungus over-winters in the twigs, and the only way to control the disease is to remove all twigs that are affected.

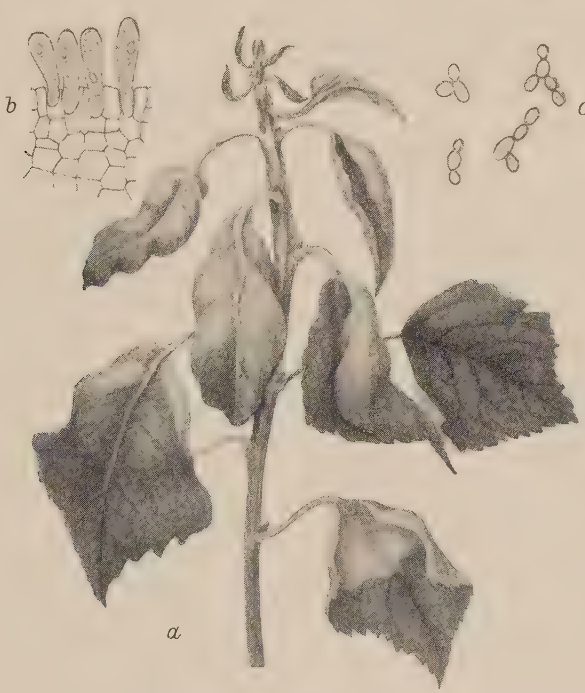

FIG. 207.—*Taphrina aurea* on *Populus pyramidalis*.
a, severely attacked tip of shoot in first year of disease ; *b*, asci ; *c*, spores budding like yeast. (Orig.)

Taphrina bullata (Berk. & Br.) Tul. produces a similar "curl" disease on the leaves of some varieties of pears and on *Cydonia japonica*. During an epidemic in the neighbourhood of Stockholm in 1885, the leaves of certain varieties of pears showed large or small, blister-like, yellow spots, often so numerous that they covered half the surface of the leaf. On the under surface the leaves were covered with a grey dust

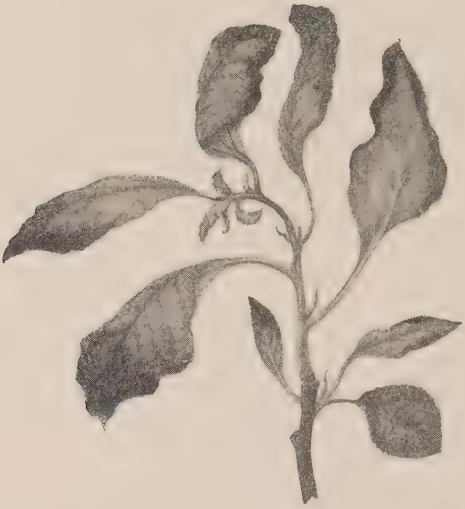

Fig. 208.—*Taphrina insititiæ* on Branch of Plum. (Orig.)

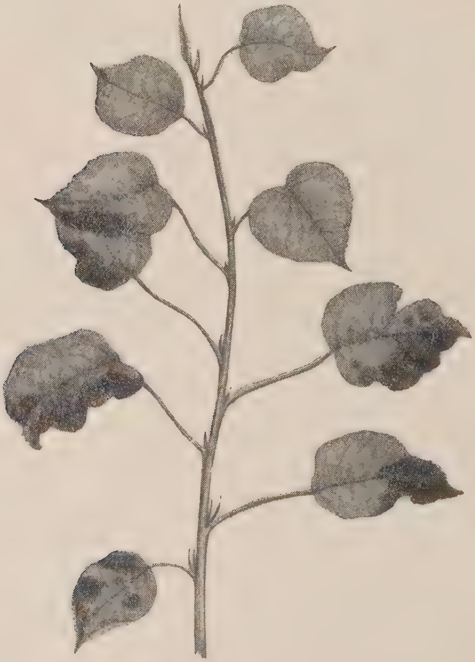

Fig. 209.—*Taphrina bullata* on Branch of Pear. (Orig.)

(ascospores). Within a few days the blisters had become black, indicating that the leaf tissue was dead; some weeks afterwards holes appeared at these places. The varieties most severely attacked were Doyenné d'hiver, Citron des Carmes, and Bodicke's Butterbirne; less severely, Beurré Diel and Königsbirne; not at all, Bellisime d'hiver. In 1923 the disease was found in the West of England on Beurré d'Amanlis (Briton Jones, 1924).

There are several forms of *Taphrina* which attack species of birch, some of the forms giving rise to witches' broom thus: **Taphrina betulina** Rostr. on *Betula odorata*, **T. nana** Joh. on *B. nana*, **T. alpina** Joh. and **T. turgida** Sad. on *B. verrucosa*. Other forms, on the contrary, produce greenish-yellow or reddish patches or blisters on the leaves, as is seen in the case of **T. Betulæ** (Fuck.) Joh. and **T. Janus** Thom. & Gries. on *B. odorata* and *B. verrucosa* and **T. carnea** Joh. on *B. odorata* and *B. nana* (Juel, 1909).

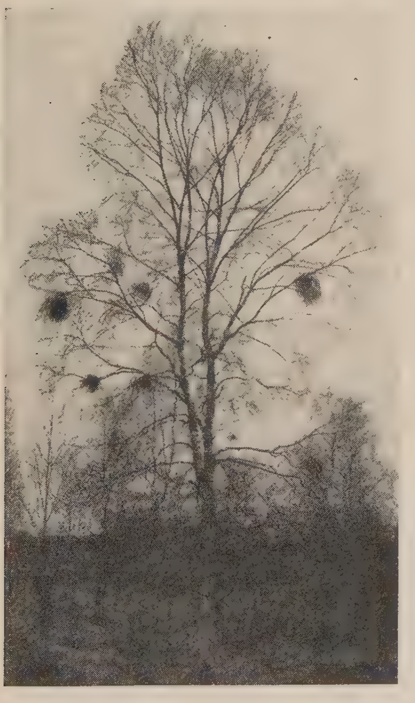

Fig. 210.—*Taphrina betulina* on Birch. Witches' brooms. (Orig.)

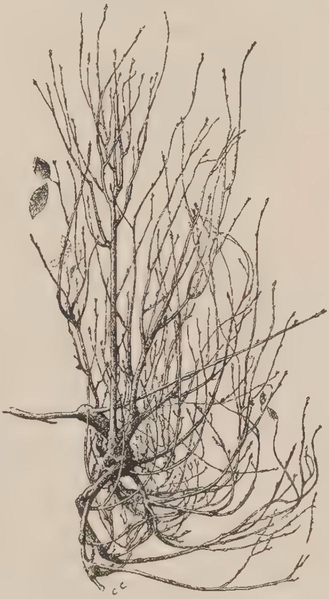

Fig. 211. — *Taphrina Cerasi* on Cherry. Witches' broom. (After E. Rostrup.)

Similar *Taphrina* diseases are found on other kinds of trees, the fungus in some cases giving rise to witches' brooms together with spotting of the leaves. The following may be noted: **Taphrina Cerasi** (Fuck.) Sadeb. on *Prunus* *avium* and *P. Cerasus*, **T. Cratægi** (Fuck.) Sadeb. on *Cratægus Oxyacantha*, and **T. Carpini** Rostr. on *Carpinus Betulus*. In other cases there is only spotting of the leaves, as **T. cœrulescens** (Desm. & Mont.) Tul. on species of *Quercus*, **T. polyspora** (Sorok.) Joh. on *Acer tartarica*, **T. Ulmi** (Fuck.) Joh. on *Ulmus montana* and **T. Potentillæ** Farl. on species of *Potentilla*.

Taphrina Tosquinetii (Wint.) Magn. produces various distortions on the young leaves and female catkins of *Alnus glutinosa* and *A. incana*. The leaves become curled and wavy; at the same time they increase considerably in size or they may have localised, blister-like swellings. A subcuticular hymenium arises on the surface of the infected leaves and out of this hymenium crowded asci grow. Later, the leaves become dry and black and fall prematurely. Some of the scales of the catkins develop into pocket-like outgrowths of varying forms (*Exoascus amentorum* Sadeb.). Young alder plants, which were inoculated in 1919 and 1920 with spore material from diseased alder leaves, showed distinct symptoms of *Taphrina* disease in 1921 on the fresh, young leaves, which indicates that the fungus can hibernate in the host plant (Klebahn, 1923).

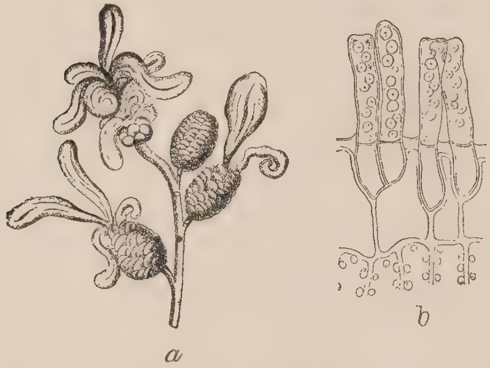

FIG. 212.—*Taphrina Tosquinetii* on Alder Catkins. *a*, deformed scales; *b*, asci. (After P. Sorauer.)

Peach Leaf Curl

Taphrina deformans (Berk.) Tul.

This disease produces distortion of the leaves of the peach tree, sometimes also of the flowers and fruit. The leaves when attacked thicken considerably and become irregularly curled and folded; they turn brown and dry and fall to the ground in summer. The flowers hypertrophy and become brittle, and whitish-green, raised areas of irregular shape appear on the fruit. The development of the flowers and the yield of fruit is very much diminished in such cases and after some weeks the trees are almost defoliated.

The fungus has an over-wintering mycelium in the bark, medulla, and medullary rays of the one-year-old twigs. From these points the mycelium grows into the new leaves which break out in the spring.

From the intercellular, branched mycelium which develops in the palisade parenchyma of the leaf there arises, on the upper surface of the latter, a hymenium of more or less closely-packed asci, each ascus with eight ascospores. In artificial cultures these spores bud like yeast cells when grown on various nutrient media and give rise to conidia, some with thin, others with thick walls; the latter are often termed resting conidia. On the host plant out of doors the ascospores generally produce short, thick germ-tubes, which give rise to conidia at the tips and also at the sides. These conidia probably can cause fresh infection on healthy parts of the plant.

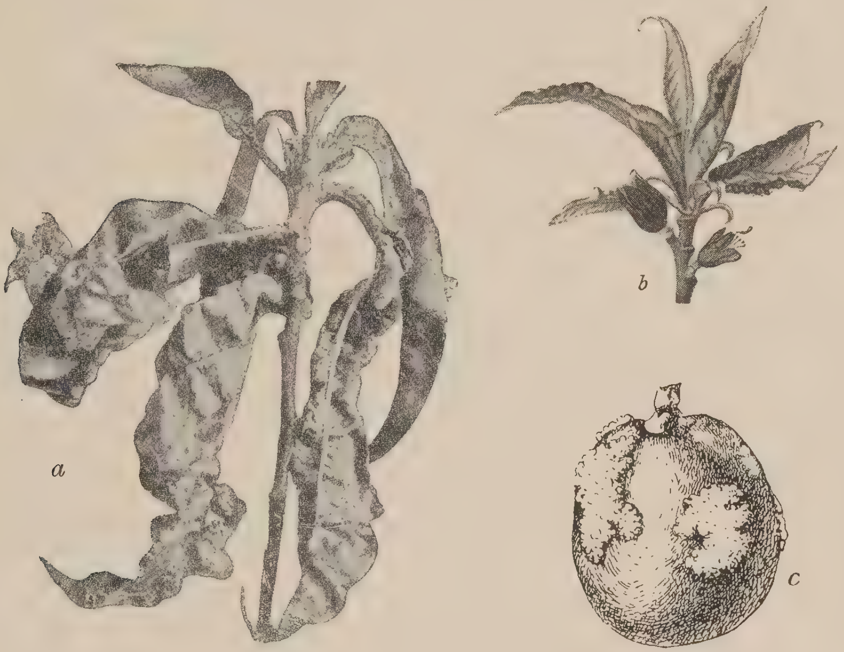

Fig. 213.—Peach Leaf Curl.

a, tip of shoot with curled leaves; *b*, diseased flowers; *c*, diseased peach. (*a*, after A. D. Selby; *b*, after M. v. Derschan; *c*, after G. Delacroix and A. Maublanc.)

Wet and cold weather during the outbreak of disease favours the growth of the fungus and at the same time the vigour of the host plant is reduced.

The different varieties of peaches vary in their susceptibility to the disease. In general, the highly cultivated late sorts are more susceptible, but susceptibility seems to vary in different districts. The disease has been found to be most severe after cold and wet springs.

The disease is now found in all peach-growing countries; it was observed in England in 1821 and in Australia in 1856. In the neighbourhood of Ithaca heavy losses occurred in 1893, 1897, and 1898. It is also known in China, Japan, and South Africa.

Control Measures.

(1) Remove all dead parts of the tree in late autumn and paint the stem with a mixture of clay, dung, and lime to which 2 per cent. fruit-tree carbolineum has been added. (2) At the same time dig the ground thoroughly and deeply and apply manure. (3) In early spring paint the stems again with carbolineum. (4) Spray the trees with colloidal sulphur (or lime sulphur), the first time when the buds begin to swell and the second time immediately before flowering. Spraying should be repeated every fortnight (in the evening in dull weather) up to three weeks before the ripening of the fruit (Winkler, 1922). In America 2 per cent. Bordeaux mixture, or Burgundy mixture of the same strength, is recommended for spraying.

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CHAPTER X

ERYSIPHACEÆ—POWDERY MILDEWS

This family, next to that of the rusts, includes the largest number of forms found in any of the groups of parasitic fungi, and many of the members cause considerable damage to cultivated plants.

The *Erysiphaceæ* attack a large number of dicotyledons belonging to different families, but amongst the monocotyledons it is only on the grasses that they are found. They occur on the leaves, particularly the upper surface, and attack young shoots, flowers, and fruits. The *Erysiphaceæ* possess a superficial mycelium which spreads over the surface of the affected organ as a delicate, hyaline, cobweb-like network, or in later stages as a dirty-grey to black felt. The mycelium sends short outgrowths (haustoria) into the underlying epidermal cells and by this means the fungus is able to withdraw nourishment from the substratum upon which it is growing. In some cases, mycelial hyphæ have been observed inside the organs, in the intercellular spaces, or even in the cells themselves.

In addition to the network of hyphæ, the mycelium produces large numbers of branched conidiophores which give rise to longer or shorter chains of conidia ; the latter are abstricted one after another and then freed. It is these conidia that give the mealy, or powdery appearance to the surface coating (mildew). The conidia, when freed from the conidiophores, are transported on any objects to which they become attached, or by wind ; they germinate very readily. When germination takes place on the young leaves or shoot of a plant which is susceptible to the fungus, the mildew appears on these parts within a few weeks.

Later on, the superficial mycelium becomes denser and more felted and develops perithecia which are visible to the naked eye as yellow, later brown to black, dots. Under the microscope these perithecia are seen to be globose or flattened and completely closed. The wall of the perithecium is formed of a layer of small, closely-fitting cells from which simple or branched outgrowths—the so-called appendages—are put out. Within the perithecium lies one ascus, or more, and each ascus encloses two, four, or eight ascospores which are unicellular and colourless. The perithecia with the enclosed asci remain unchanged through the winter, but in spring they rupture and the spores are expelled. When the ascospores germinate on a suitable host they give rise to fresh outbreaks of disease.

In certain cases the perithecia, which are designed for the over-wintering of the fungus, are not formed. This may occur year after year over large districts, but in spite of the absence of perithecia the disease may reappear annually. The hibernation of the mycelium is regarded as the means by which the fungus persists, but it is also possible that it continues within the cell tissues of the diseased parts in a form which it is difficult to observe.

The different species of the genus are generally characterized by the appearance of the appendages, especially by the manner in which they branch, also by the number of asci in each perithecium and the number of ascospores in the ascus. Within each species, just as in the case of the rust fungi, there are often several biologically different, " specialised " forms which are confined to one or more host plants.

The injury caused by the mildew fungi is partly due to the covering of the affected part by the mycelium, as in this way light and air are kept from the plant, partly, also, to the removal of food by the haustoria which penetrate the epidermal cells. The diseased parts of the plant often wilt prematurely and die.

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Erysiphe

The perithecia contain numerous (8–20) asci and each ascus 8–12 spores. The appendages are simple or slightly branched, in general, similar to the ordinary hyphæ.

Mildew of Cereals and Grasses

Erysiphe graminis DC.

The mycelium produces a more or less extensive, cobweb-like growth, white or pale red in colour, on the sheaths and blades of the leaves. Some of the hyphæ grow out at right angles and abstrict chains of conidia which germinate easily and so spread the disease. This stage in the development of the fungus appears, not infrequently, early in the spring on varieties of autumn-sown cereals. The fungus has then a reddish-grey appearance and is sometimes mistaken for rust. If the weather be favourable to the development of the fungus it passes into a greyish-white dense felt in which numerous small, brown or black perithecia are embedded. A few weeks after the first appearance of the perithecia, the leaves which have been covered with the mildew fungus die and finally the felt-like covering falls off. It is by means of the perithecia that the fungus persists during the winter time. The effect of the fungus is to cause the plants to ripen prematurely and the seeds to remain small and shrivelled. In some years the disease gives rise to very serious havoc in the wheat crop.

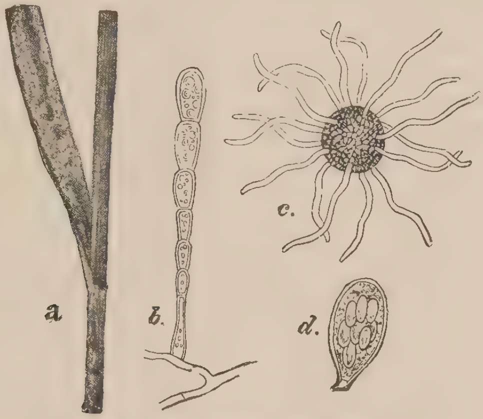

FIG. 214.—Mildew on Wheat.

a, wheat haulm with coating of fungus ; *b*, chain of conidia ; *c*, perithecium with appendages ; *d*, ascus with eight spores. (*a*, Orig. ; *b–d*, after E. Rostrup.)

A number of specialised forms of this species of fungus are known : (1) f. sp. Tritici on *Triticum vulgare*. *T. spelta*, *T. polonicum*, and *T. turgidum* ; (2) f. sp. Hordei on *Hordeum hexastichum*, *H. vulgare*, *H. trifurcatum*, *H. nudum*, *H. jubatum*, and *H. murinum* ; (3) f. sp. Secalis on *Secale cereale* and *S. anatolicum* ; (4) f. sp. Avenæ on *Avena sativa*, *A. fatua*, *A. orientalis*, and *Arrhenatherum elatius* ; (5) f. sp. Poæ on *Poa annua*, *P. trivialis*, *P. pratensis*, *P. cæsia*, *P. nemoralis*, and *P. serotina* ; (6) f. sp. Agropyri on species of *Agropyrum* ; and (7) f. sp. Bromi on species of *Bromus*, preferably on *B. mollis* and *B. sterilis*.

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Erysiphe communis Grev. forms a thin, white, regularly-spread membrane with numerous black perithecia on the leaves and stems of a number of plants such as rape, pea, vetch, lupine, etc. In this collective species there are a number of specialised forms : Group A : (1) f. sp. Trifolii repentis on *T. repens ;* (2) f. sp. Trifolii hybridi on *T. hybridum ;* (3) f. sp. Trifolii pratensis on *T. pratense* and *T. medium ;* (4) f. sp. Trifolii agrarii on *T. agrarium ;* Group B : (1) f. sp. Medicaginis sativæ on *M. sativa* and *M. falcata ;* (2) f. sp. Medicaginis lupulinæ on *M. lupulina ;* Group C : (1) f. sp. Lathyri montani on *L. montanus ;* (2) f. sp. Lathyri pratensis on *L. pratensis ;* Group D : f. sp. Pisi on *P. sativum* and *P. arvense ;* Group E : f. sp. Viciæ sativæ on *V. silvatica*, *V. sepium* and *V. sativa ;* Group F : f. sp. Loti on *L. corniculatus* and *L. uliginosus ;* Group G : f. sp. Meliloti on *M. Petitpierreanus*, *M. altissimus* and *M. albus ;* Group H : f. sp. Brassicæ on *B. Napus*, *B. Rapa*, and *B. oleracea* ; and Group I : f. sp. Pastinacæ on *P. sativa*. Altogether, including wild species of host plants, twenty-six specialised forms of this species have been distinguished (Hammarlund, 1924).

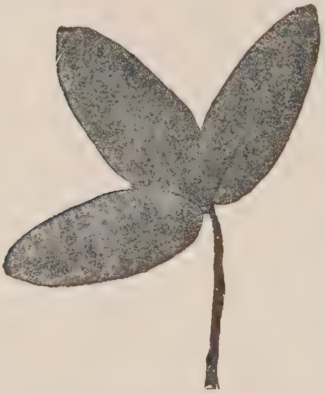

Fig. 215.—Mildew on Clover. Leaf of *Trifolium medium*. (Orig.)

E. Cichoriacearum DC., also named *E. lamprocarpa* Kickx., *E. Linkii* Lév., etc., attacks many of the *Compositæ* (chicory, sunflower, aster), *Cucurbitaceæ*, etc. Here also there is specialization : one form on

Cucurbitaceæ (on at least seventeen species belonging to ten genera), another on the aster, and a third on *Solidago*, etc. (Reed, 1907, 1908).

E. Acaciæ Eriks. sometimes appears on *Acacia lophantha* in glasshouses. The fungus first attacks the older leaves and gradually spreads upwards. The leaves lose their colour and drop; both surfaces are covered with a felt of fungus in which perithecia in large numbers occur. The disease appeared annually in a glasshouse near to Stockholm in the eighties of the last century and caused much damage; the perithecia ripened in February (Eriksson, 1885).

Control Measures, against mildews in general:

(1) Seed should be chosen from plants that have been free of mildew. (2) Stems and leaves of plants on which perithecia are found in considerable quantities should not be thrown on the dung- or compost-heap.

Uncinula

The perithecia contain numerous asci and each ascus eight ascospores. The appendages of the perithecia are almost always hooked at the tip or spirally bent.

Vine Powdery Mildew

Uncinula necator (Schw.) Burr.

Oidium Tuckeri Berk.

This fungus forms a greyish- to silver-white network or powder on the green parts (stem, leaf, inflorescences, fruit) of the vine. At first the fungus is found in scattered patches, but these gradually coalesce until all the parts mentioned are covered. The network consists of hyphæ which in some places send out short, branched haustoria and in others long, erect, unbranched conidiophores. The conidia germinate quickly and, within a few days, are able to give rise to new centres of infection. The leaves when attacked wilt prematurely. In damp weather the grapes crack and decay, in dry weather they may ripen but they remain hard and sour and not fit to eat.

This fungus, which has been known in America for nearly ninety-five years (Schweinitz, 1834), was discovered in Europe some eleven years later by Tucker at Margate (South of England) and two years later was recorded in European literature under the name of *Oidium Tuckeri* (Berkeley, 1847). In France (Versailles) the fungus appeared in vineries in 1848 and 1849, but not until 1851 in the French vineyards. Within a few years it had spread to all grape-growing countries of Southern Europe, being especially prevalent in Spain, Italy, and Greece, as well as in Mediterranean countries outside Europe, such as Algeria, Syria, and Asia Minor. In 1852 the disease was observed in Madeira and in 1866 in Australia.

An outbreak, which resulted in the loss of all the grapes, occurred in a vinery near to Rosenborg in 1857 and is the first record of the disease in Denmark. The first outbreak in Sweden was one which occurred in 1905 at Börringekloster in Scania, on vines imported from England.

The devastating spread of the fungus in Southern Europe in the years 1852–55 caused much anxiety and led to the formation of scientific commissions to study the disease and find methods for its control. In some places, such as the neighbourhood of Montpellier (South of France), the destruction of the crop was sometimes as much as nine-tenths of the whole. In other places, especially in Italy, the growing of grapes began to be regarded as impossible and the labourers migrated in large numbers to America.

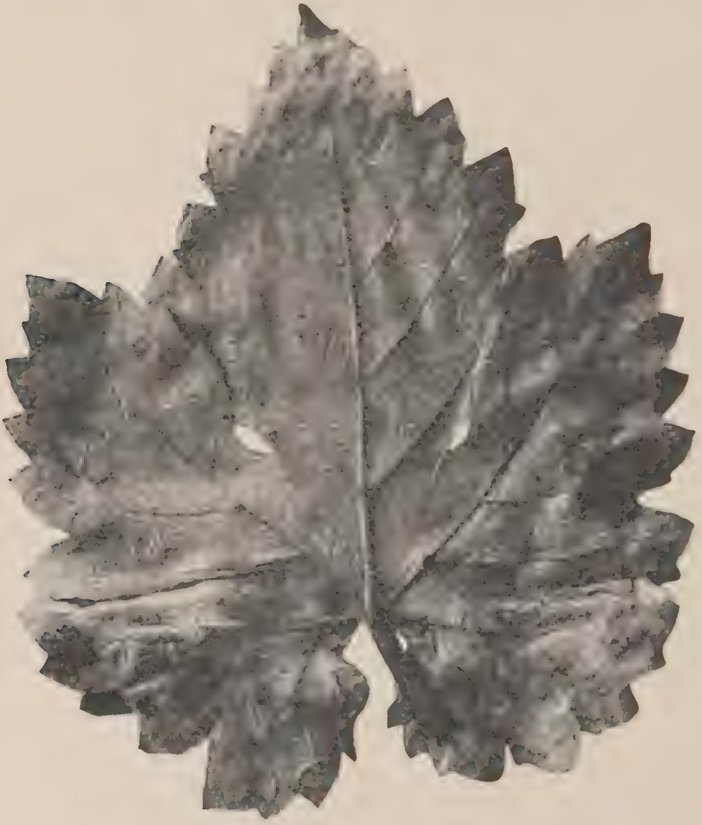

Fig. 216.—Vine Powdery Mildew. Conidial stage (*Oidium Tuckeri*). (Orig.)

A perithecial stage of the fungus regularly follows the white conidial stage in America, but in Europe this only occurs in exceptional cases. The perithecia are found mainly on the upper surface of the leaf, more rarely on the under surface or on the inflorescences, as very small, black dots scattered over a grey felt. The appendages are spirally curled at the tips.

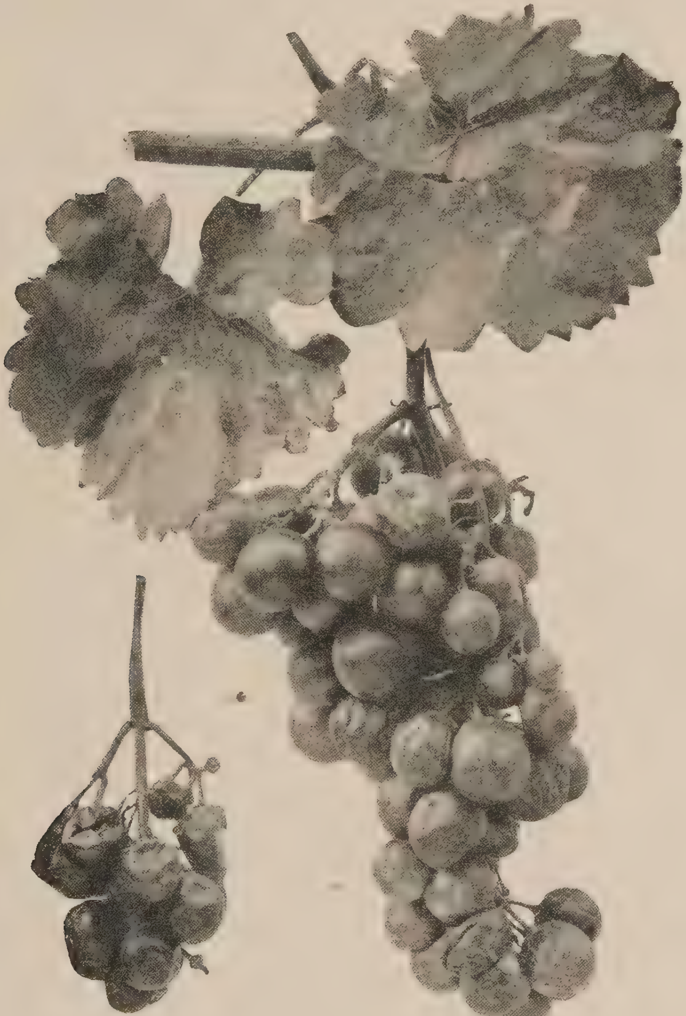

FIG. 217.—Vine Powdery Mildew. Conidial stage (*Oidium Tuckeri*). (Orig.)

For a long time it was not known how the fungus continued to live from year to year in European vineries and vineyards as the perithecia, which contain the winter spores, only rarely developed there. It was not until 1892–93 (forty-seven years after the first appearance of the conidial stage in Europe) that perithecia were discovered in France (Couderc, 1892; Viala, 1893; Prillieux, 1893). Since that time the perithecial stage has only seldom been detected (in Bulgaria, 1923), although the fungus in its white, powdery form appeared to a large extent each year.

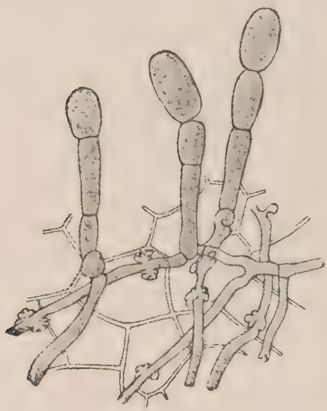

Fig. 218.—Vine Powdery Mildew. Conidial stage (*Oidium Tuckeri*). Conidiophores. (After O. Kirchner and Boltshauser.)

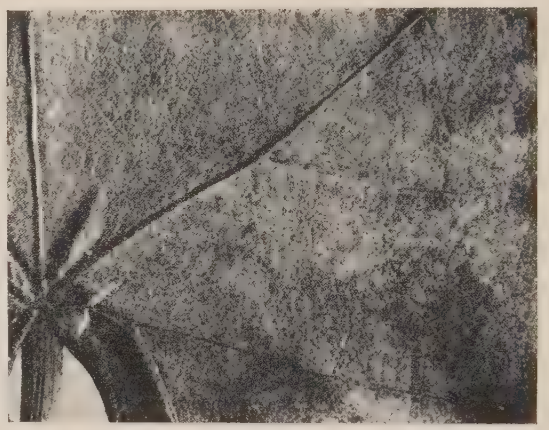

Fig. 219.—Vine Powdery Mildew. Perithecial stage (*Uncinula necator*). Portion of leaf seen from below. (After G. P. Clinton.)

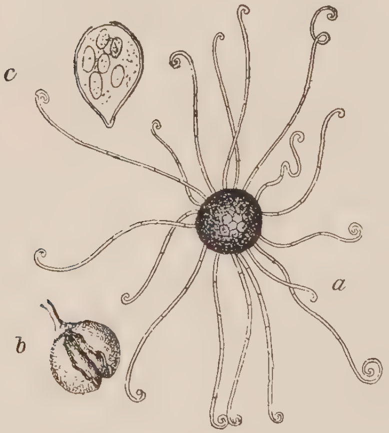

Fig. 220. — Vine Powdery Mildew. Perithecial stage (*Uncinula necator*).

a, perithecium with appendages; *b*, burst grape; *c*, ascus. (After G. Delacroix and A. Maublanc.)

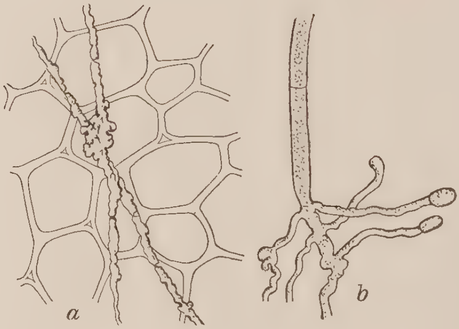

Fig. 221.—Vine Powdery Mildew. Overwintering mycelial stage.

a, knotted swollen hyphæ; *b*, formation of conidia from the hyphæ. (After G. de Istvánffi.)

It became necessary, therefore, to look for some other way in which the fungus might hibernate, and attention was given to the possibility of the mycelium persisting in the stem and winter buds.

In Germany in November, 1903, certain short, nodular hyphæ were discovered on the reddish-brown patches which had been produced on one-year shoots by the *Oidium* stage of the fungus during the previous growing period. In the following spring typical conidiophores developed from these hyphæ and produced conidia (Appel, 1903); a similar discovery was made about the same time in Hungary. Shoots which had had a thick coating of white mildew in autumn were kept under observation from October 11, 1903, to February 13 of the following year. This led to the discovery of a persistent mycelium, not only on parts of the stem, which at first sight appeared almost uninjured, but also in the winter buds, the corky tissue of the shoots, and in the shrivelled flesh of the grapes. The mycelium on the stem was in the form of knotted swellings which germinated normally in artificial cultures, whilst the mycelium in the shoots was found in the intercellular spaces as a network of dark yellow hyphæ filled with protoplasm (de Istvánffi, 1904).

The American vines, which are descendants of *Vitis labrusca*, *V. rupestris*, *V. riparia*, *V. Berlandieri*, etc., suffer, in general, less than the European varieties which are descended, mainly, from *V. vinifera*. Some European vines, such as Traminer and Riessling, are relatively resistant, whilst others, such as Frankenthaler, Muskateller, Malvasier, etc., suffer considerably. In Hungary, low temperatures in May and June, a low water-vapour pressure in the atmosphere, and also west and south-west winds in April and June, appear to lead to severe outbreaks of mildew (Sajo, 1901).

Dusting the vines with sulphur, either flowers of sulphur or finely-ground sulphur, is the best means of dealing with the disease; sulphur precipitated from liver of sulphur is also helpful. Special machines have been designed for dusting and by means of them the sulphur can be well distributed. Sulphuring should be carried out in the middle of the day by sunshine and not early in the morning when the vines are still wet with dew.

Protective Measures :

A. In vineries. (1) In late autumn, immediately after the grapes have been gathered, all remains of grapes and shoots should be removed and destroyed if disease has occurred in the vinery during the growing season. The vines as well as the walls, roof, and soil should then be thoroughly sprayed with a solution of liver of sulphur (1 oz. to 6 galls. water). (2) Early in spring, immediately before the buds open, all young shoots should be painted with milk of lime. (3) If traces of mildew are visible in spring the parts that are affected should be sprayed with a solution of liver of sulphur or else dusted with sulphur, and the treatment repeated every second week, when necessary. B. In vineyards. (4) The vines should be dusted with sulphur on the first appearance of the disease, again during flowering and, if necessary, a third time one or two weeks later. C. In all circumstances. (5) When purchasing new vines for planting, a guarantee should be obtained that mildew has not occurred in the nursery, from which the plants are obtained, during the past 2–3 years.

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Mildew of Maple, *Uncinula Aceris* (DC.) Sacc. and *U. Tulasnii* Fuck., attacks the leaves of maples (*Acer platanoides*, *A. pseudoplatanus*, *A. campestre*, etc.). Both species of the mildew often appear together on the same plant. *U. Aceris* produces a delicate, filamentous mycelium on the young leaves, especially those of *Acer platanoides*, and causes them to become greenish-yellow and curled. *U. Tulasnii* is found on fully developed leaves, always on the under side, and produces pure white patches on the otherwise green surface ; the patches extend until the upper surface of the leaf becomes almost entirely white. The two species are morphologically different in that *U. aceris* has rectangular-oval conidia 20 μ long and 18 μ broad, whilst those of *U. Tulasnii* are spherical and only 8 μ in diameter. The latter species has sometimes caused considerable damage, especially on young plants in the nursery, in the neighbourhood of Stockholm. Black perithecia are found in groups embedded in the white felt. As in this form of mildew the perithecia occur only on the leaves, it is probable that if the fallen leaves were collected in autumn and burnt, an outbreak of disease in the following spring would be prevented (Eriksson, 1885).

Willow mildew, *Uncinula Salicis* (DC.) Wint. produces scattered, circular, white patches of mildew, in which perithecia are embedded, on the leaves of species of *Salix* and *Populus ;* the leaves often fall prematurely.

Podosphæra

The perithecium contains only one ascus with about eight spores. The basal appendages of the perithecium are short, the apical appendages very long and not branched.

Powdery Mildew of Apple

Podosphæra leucotricha (Ell. & Ewerh.) Salm.

In this disease the leaves and leaf-stalks, especially those at the tips of the terminal shoots, are seen to be thickly covered with a white, flour-like powder. The same powdery appearance is also seen on the flower stalks and sepals. The blossoms, together with the stalks, become withered and dry and fall prematurely. Soon afterwards the leaves of the affected one-year-old shoots die and the shoots become almost completely defoliated. From the surface mycelium short erect branches arise (*Oidium farinosum*) which at their tips abstrict unicellular, colourless conidia, and by means of the latter the disease is disseminated. When the conditions are favourable to the fungus, it produces brown,

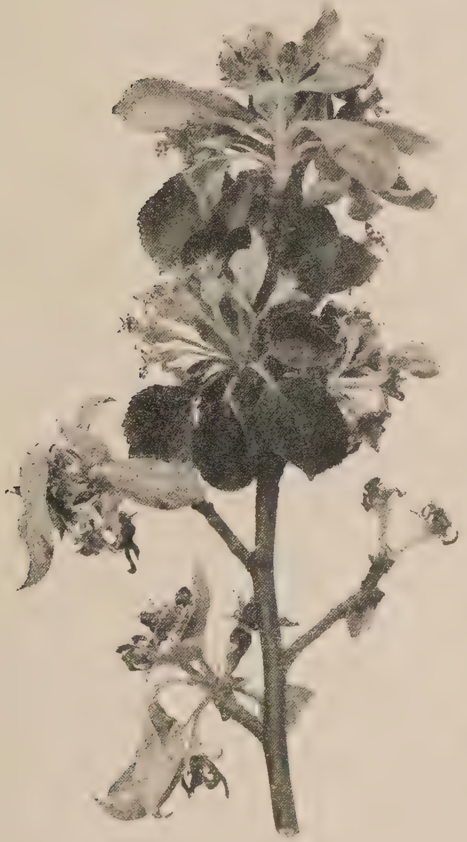

FIG. 222.—Apple Mildew. Flower trees attacked by mildew. Oidium stage (*Oidium farinosum*). (Orig.)

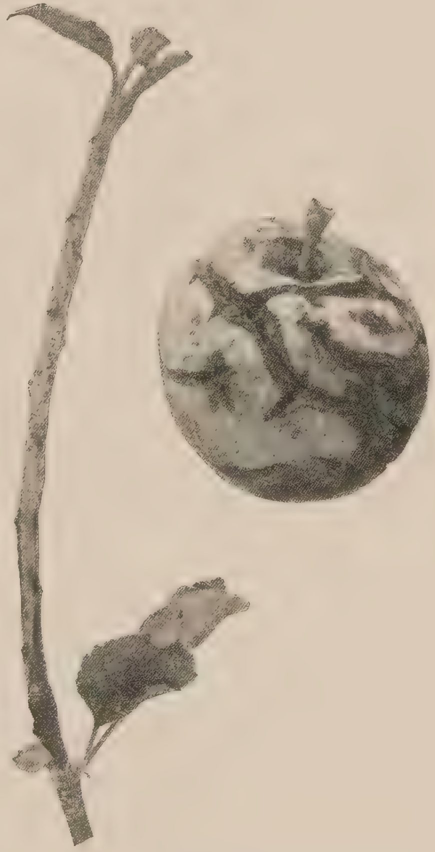

FIG. 223.—Apple Mildew. Annual shoot and apple attacked by mildew. Perithecial stage. (Orig.)

dot-like perithecia with filamentous appendages and it is by means of these perithecia that the fungus is able to over-winter. The lower appendages are short and tortuous and of a brown colour, the upper ones straight, rigid, and hair-like, the lower portion brown, the upper portion colourless.

This disease causes most of the fruit to fall prematurely; the few apples that remain on the tree are spoilt in appearance; on the skin is a network of hyphæ with here and there groups of perithecia embedded in it. Deep cracks crossing one another are formed in the flesh of the fruit. In severe outbreaks the loss of fruit may be 25–75 per cent. of the crop.

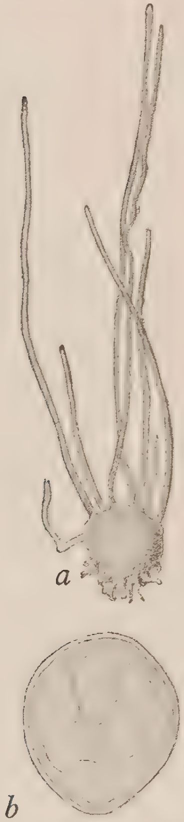

Fig. 224.—Apple Mildew.
a, perithecium with appendages; *b*, ascus. (After P. Magnus.)

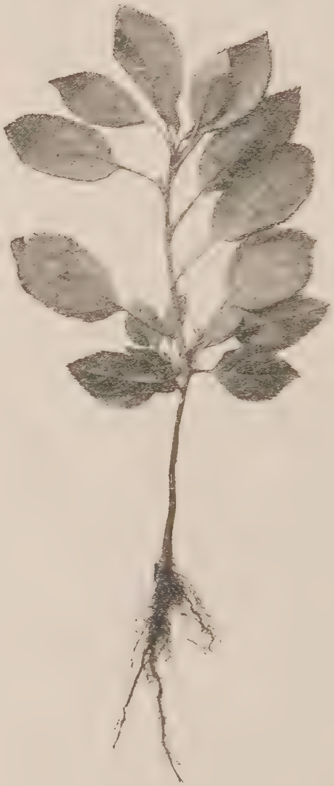

Fig. 225.—Apple Mildew. Apple seedling completely covered with Oidium stage of fungus. (Orig.)

Frequently, very severe attacks of the fungus, but only in the conidial stage, have been observed on young apple seedlings. In one case, north of Washington, a nursery containing 400,000 stocks and grafted trees suffered from the disease; the leaves were at first covered with a greyish-white powder and gradually became dry and brittle; the disease affected the stocks so severely that they were in many cases too weak to be grafted (Galloway, 1889).

The conidial stage of the disease was seen at the beginning of August, 1906, in a nursery near to Stockholm, on thousands of young apple seedlings grown from imported seeds. As the disease had not previously occurred in the nursery and was not found on other apple trees it must be assumed that the fungus was introduced on the imported seeds. It was noticed that the badly infected leaves fell prematurely. The course of the disease was carefully followed up to the beginning of winter, but no trace of perithecia was discovered; the diseased seedlings were not sprayed or given any other treatment. In the following years, 1907–08, no signs of mildew were observed on the stocks from the diseased plots nor on older trees in the garden.

Latterly, other observations have suggested that the fungus can hibernate vegetatively in the stem tissues of diseased shoots. Apple twigs which were severely attacked by mildew in the summer of 1921 were treated in January, 1922, first by dipping in warm water (35° C.), then sprayed with various fungicides and finally placed in a greenhouse where the probability of fresh infection was very slight. When the buds opened most of the leaf structures were white with mildew. The fungus appeared to have spread upwards from the base of the leaf-stalk to the tip of the leaf (Höstermann, 1922; Foëx, 1923).

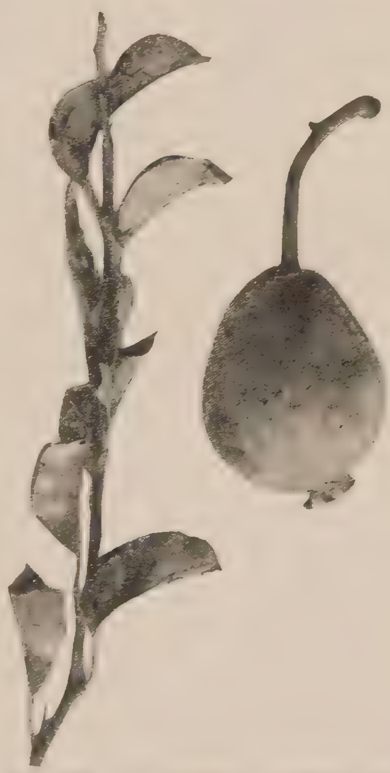

FIG. 226.—Annual Shoot and Fruit of Pear Tree attacked by Apple Mildew. (Orig.)

Sometimes the fungus attacks pear trees growing in the same garden as apple trees which are suffering from an outbreak of mildew.

The fungus was first described from America in 1888, in 1894 it made its appearance in Europe (Tyrol), about the same date it was observed in Russia, Japan, and Australia, and in 1906 in Sweden. It is now known in all apple-growing countries and in many districts it is regarded as one of the diseases which give rise to heavy losses in fruit-growing.

Control Measures :

(1) Diseased one-year-old shoots and leaves should be removed in autumn and burnt. (2) When the trees are bare they should be sprayed with 1 per cent. solutions of copper sulphate or liver of sulphur and the ground under the trees should be limed. (3) Spray the trees three or four times in spring with lime sulphur, colloidal sulphur, or Solbar (*a*) just before the petals fall, (*b*) immediately after the fruit has set, (*c*) two weeks later, (*d*) again two weeks later. (4) Seeds to be used for raising seedlings should be obtained from a garden where mildew is known not to have occurred.

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Podosphæra Oxyacanthæ (DC.) de By. In some years this fungus causes havoc to *Cratægus Oxyacantha*, *C. monogyna*, etc., growing in nurseries. It gives rise, at first, to reddish-white patches on the terminal leaves which are badly curled, and these patches extend until the leaves are almost wholly covered. After some weeks, brownish-black perithecia appear in the white mycelial covering on the leaves and to a lesser degree in that on the stems. Many plants are killed by the fungus. In some years perithecia are not formed (Eriksson, 1885 ; Maurizio, 1927).

The mildews which occur on species of *Amelanchier*, *Prunus*, *Sorbus*, *Spiræa*, and *Vaccinium* are usually regarded as a variety of *P. Oxyacanthæ* (Salmon, 1900), but according to more recent investigations the form on *Cratægus*, *Cydonia*, and *Sorbus* appears in each case to be a biologically independent sub-species (E. Fischer, 1926).

Sphærotheca

The perithecium contains only one ascus which has eight spores. The appendages are simple and only placed basally.

Rose Mildew

Sphærotheca pannosa (Wallr.) Lév.

This fungus attacks the leaves, young shoots, and flowers of rose trees growing in the open and in the greenhouse. On the upper surface of the fresh, green leaves, dull white patches appear and on the under surface an extended, white coating. Within a few days the leaves become covered with a white powder due to the formation of conidia (*Oidium leucoconium* Desm.). The hyphæ cover the tips of the one-year-old shoots and cause them and their leaves to curl. The fungus frequently does not progress beyond this conidial stage—especially in the greenhouse. Later in the season, out-of-doors, the stems and flower buds are usually covered with a dirty-black, felted mycelium in which black perithecia are embedded. This felt persists into the late autumn, or even winter, and by means of it the fungus hibernates. In cases where there are no perithecia and no autumn mycelium, especially where the disease reappears each year in the greenhouse, the hibernation of the fungus must be considered to be due to the persistence of the mycelium in the peripheral tissues of the stem, although proof of this has not yet been obtained. The fungus does more damage to out-of-door roses in dry and hot summers, whilst in the greenhouse, sudden changes of temperature, ventilation, drying out of the soil, all appear to favour the

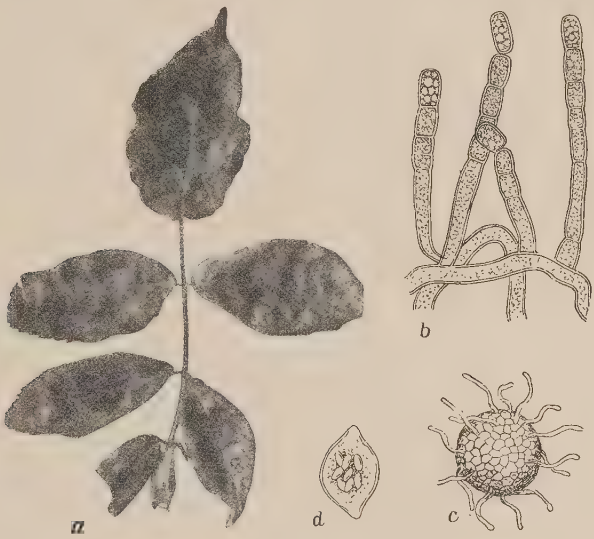

FIG. 227.—Rose Mildew.

a, diseased leaf; *b*, conidiophores; *c*, perithecium; *d*, ascus. (*a*, after E. M. Freeman; *b*, Orig.; *c* and *d*, after F. Ferraris.)

disease. When a greenhouse is no longer regularly heated, care should be taken to maintain a temperature of not less than 15°–18° C. (say 60°–65° F.) in the evening and 10° C. (50° F.) at night; further, the leaves should be dry over-night (Schultheis, 1898).

Roses vary in their susceptibility to the disease, the well-known variety Crimson Rambler being often severely attacked, particularly if grown on a warm south or south-west wall and where water from the roof drips on to it (Sorauer, 1915). Cutting the plant down to the ground in autumn was found to have good results in the South of Sweden, the new shoots in the following spring being practically free from disease. Spraying with a tar emulsion has been recommended from Italy, but there is risk of spoiling the flowers (Guercio and Baroni, 1895).

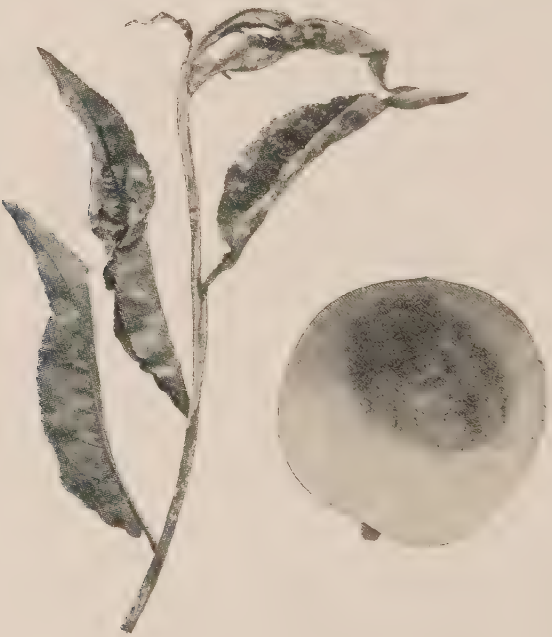

Fig. 228.—*Sphærotheca Persicæ.* (Orig.)

The fungus was first reported from Germany in 1819 under the name *Alphitomorpha pannosa* (Wallroth) and in Sweden at the beginning of the forties of the last century; it is now found in all countries where roses are grown. The connection between the conidial and perithecial forms was proved in 1851.

Control Measures:

(1) Greenhouse roses should be frequently dusted with flowers of sulphur or ground sulphur. (2) Roses out of doors, when badly affected

with the perithecial felt, should be cut down drastically, even to the ground.

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Sphærotheca Persicæ Woronich. sometimes causes considerable injury to peach trees (*Persica vulgaris*). The young shoots are covered with a white powder and the leaves are curled and folded together. Light patches, becoming darker, appear on the fruits, which cease to grow and become inedible.

The fungus was at first considered to be identical with that of the rose mildew, but inoculation experiments made in Sweden in 1906 with conidial material from the rose on to the tender buds and leaves of the peach were unsuccessful, and a similar result was obtained in France in 1914. These results led to the discovery of certain differences in the size of the spores of the two forms of mildew (Woronichin, 1914).

The best protective measure appears to be repeated dusting with flowers of sulphur.

American Gooseberry Mildew

Sphærotheca mors-uvæ (Schw.) Berk.

This fungus attacks the gooseberry (*Ribes Grossularia*), appearing on the young berries when they are about the size of a pea, and on the young leaves and stems, not, however, on the leaves of the older parts of the stem, as a delicate white network of hyphæ. From this network, isolated erect branches arise which produce conidia in long chains; the conidia give a floury appearance to the parts of the plant that are affected. It is by means of these conidia that the disease is easily and rapidly spread to neighbouring plants.

Three different outbreaks of the disease have been distinguished. (1) The spring attack (in June) on the young berries, often also on the young shoots. The origin of this outbreak is the external infection from resting mycelium on the same bush or on a neighbouring one. (2) The summer outbreak (July) on young, newly-planted, strongly-growing bushes and on vigorous new shoots of older bushes which have been cut down to the ground in the previous autumn. This outbreak is due either to external infection or to a hidden source of disease (mycelium or plasm) in the plant. (3) The autumn outbreak (August to October), which is to be ascribed mainly to infection from other diseased bushes.

The tips of the shoots attacked in early spring do not ripen but die back, the shoot sometimes being distorted and the twisted tip standing out bare and hollow.

The snow-white mycelium changes after 8–14 days to a chocolate-brown to black colour and develops into a thick felt which, partly or wholly, covers the branches as well as the berries. The berries do not, as a rule, cease to grow, but remain on the bush until harvest-time; their appearance is, however, spoilt and they are not fit for use. Numerous black, dot-like perithecia, which are spherical and provided with long, simple appendages, occur embedded in the mycelial felt. Each perithecium contains a single ascus with eight spores. The fungus over-winters by means of the perithecia, and there is no doubt the outbreaks of disease in the spring arise from this source. Observations made in England in 1914 indicate that the latter source of infection is not the only one, as the careful examination, from February to May, of parts of the stem which were covered by the mycelium failed to show any

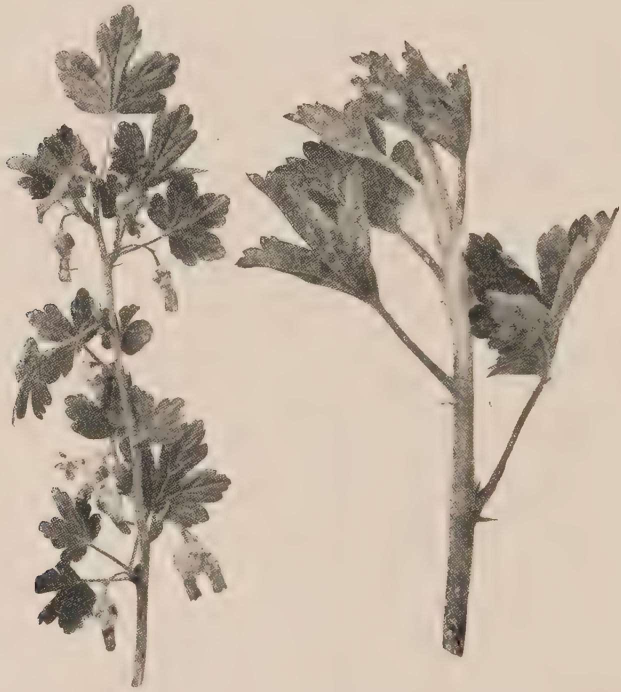

Fig. 229.—American Gooseberry Mildew in early, powdery Oidium stage. Left, on developing berries; right, on leaves of annual shoot. (Orig.)

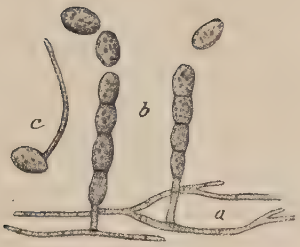

FIG. 230.—American Gooseberry Mildew. Conidiophores and conidia. (Orig.)

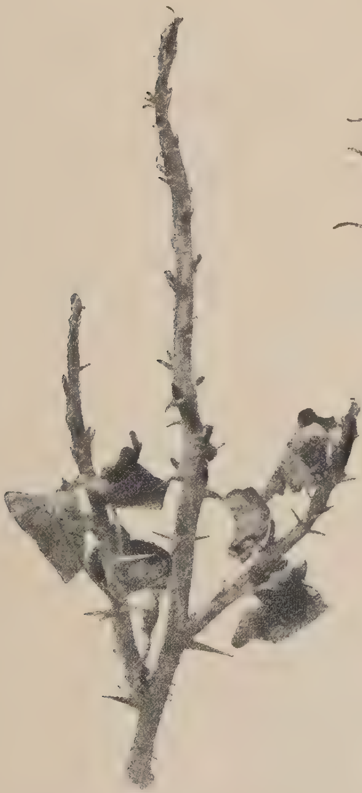

FIG. 231.—American Gooseberry Mildew. Gooseberry shoot with severe attack, partially defoliated and swollen. (Orig.)

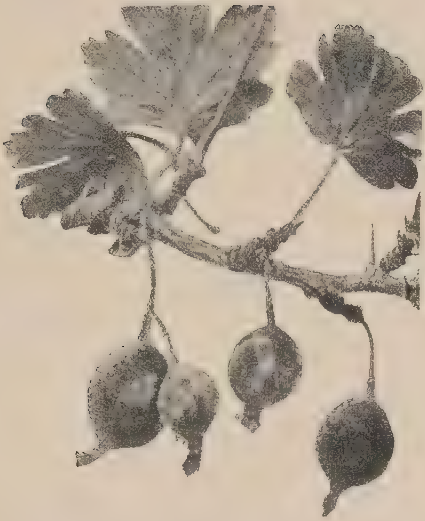

FIG. 232.—American Gooseberry Mildew. The black perithecial stage. (Orig.)

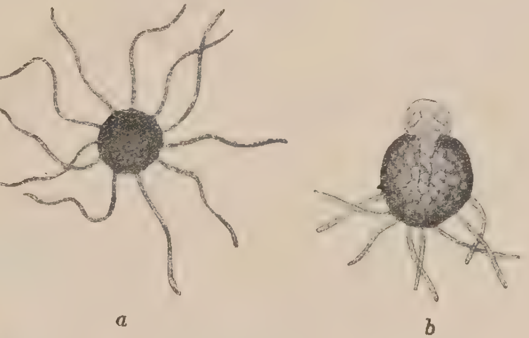

FIG. 233.—American Gooseberry Mildew. *a*, perithecium ; *b*, emerging ascus. (Orig.)

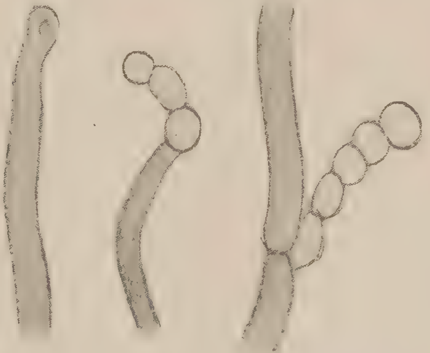

FIG. 234.—American Gooseberry Mildew. Over-wintered mycelium germinating in February. (Orig.)

perithecia or spores in a viable condition (Salmon). Further, it was also noticed in February of one year, near to Stockholm, that there was a direct germination of the over-wintered mycelial hyphæ.

The summer outbreak at the middle or end of June cannot always be explained by a hibernating mycelium or by infection from neighbouring centres of conidia. Numerous exact observations made in different parts of Sweden during the years 1905–08 lead to the conclusion that the fungus lives within the diseased branches or stem of the gooseberry bush in a latent form, and from these points the infection spreads. It is conceivable that in late autumn, at the end of the growing period, the sap, infected by the fungus, passes to the stem and roots, rising again in the following spring, when conditions are favourable and the annual shoots are at a certain stage of development, to produce a fresh summer outbreak of the disease. Indications of an internally diseased state of the cortical and medullary tissues of the young annual shoots of a gooseberry bush, which had suffered from a severe attack of mildew in the previous year, were afforded by the differences which were observed in the anatomical structure of the shoots from this diseased bush and those from a healthy one. In cross-section the diseased shoots showed more protoplasm in the cells and a larger accumulation of tannic acid than was the case in the healthy shoots. To what extent and in what form a vegetative stage of the fungus may exist in the tissues has not yet been investigated.

In the course of the winter the brownish-black mycelium on the stem of the shoot changes to an ashen-grey colour. After some years the gooseberry bushes become stunted and misshapen owing to the continued dying-back of the new shoots.

The chief host plant is *Ribes Grossularia*, and in Europe the fungus has also been observed on *R. nigrum*, *R. rubrum*, *R. aureum*, *R. alpinum*, and *R. atropurpureum*. In North America it has been found on *R. cereum*, *R. Cynosbati*, *R. divaricatum* var. *irriguum*, *R. floridanum*, *R. gracile*, *R. Hudsonianum*, *R. lacustre*, *R. missouriensis*, *R. prostratum*, and *R. rotundifolium*.

On red and black currants the fungus appears on the under side of the younger leaves, especially in the angles of the veins near the midrib, as a light-coloured felt and on the berries as black cushion-like patches.

In the case of *Ribes aureum* the diseased tips of the shoots are much deformed and are completely covered with a white powder.

It was noticed in the South of Sweden (Bjerred near Lund) that raspberries growing near to diseased gooseberries and black currants also suffered from a severe outbreak of mildew, both on the leaves and berries, in the years 1906–08, but no perithecia were to be found.

The American gooseberry mildew has been known in America since 1834. The outbreaks in that country have been so severe that the growing of gooseberries, especially the large-berried European varieties which are all descendants of *Ribes Grossularia*, has to a large extent

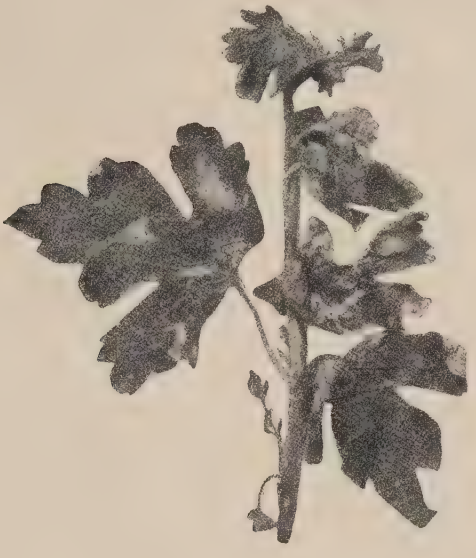

Fig. 235.—*Ribes aureum* attacked by American Gooseberry Mildew. (Orig.)

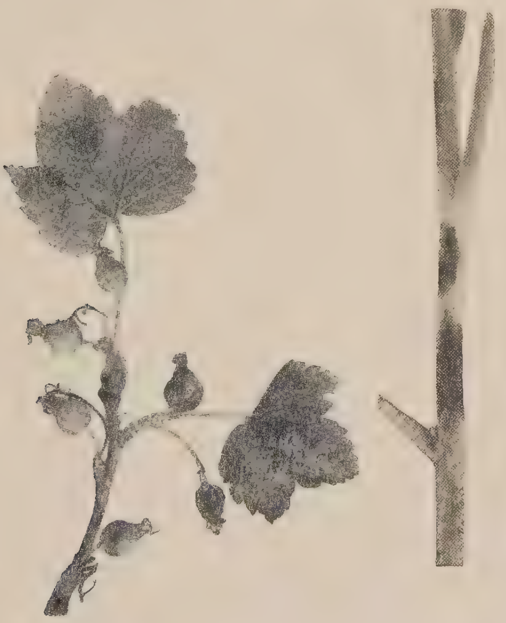

Fig. 236.—*Ribes nigrum*, attacked on berries and internodes of branches by American Gooseberry Mildew. (Orig.)

been given up. Varieties obtained by crossing European and native sorts—the latter are more resistant as a rule—appear, up to now, to be less susceptible to the disease. The fungus reached Europe in two ways —east and west; the introduction from the east appears to have been into South-West Russia (Winnitzy) at the end of the eighties of last century on American gooseberry bushes. In 1904 the disease was established in Russia and spread to the neighbouring countries in Eastern Europe (Regel, 1907). The western introduction was about 1900 to Ireland and Denmark, and from these countries the disease has spread to all parts where gooseberries are grown.

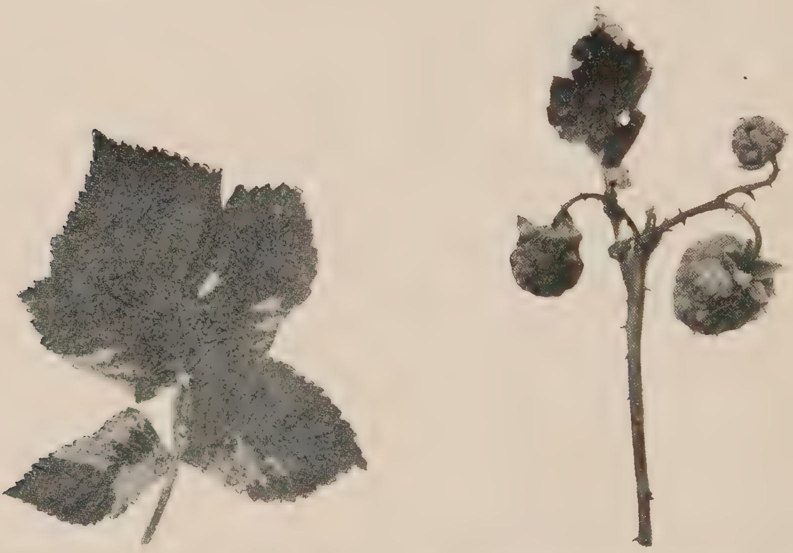

FIG. 237.—*Rubus Idæus*, attacked on leaves and fruits by American Gooseberry Mildew. (Orig.)

Where the disease has gained a footing it has usually spread in spite of attempts to stamp it out. Spraying with different fungicides and at various times of the year has been practised, as has also the removal of infected shoots even to cutting down the bushes to the ground, and this in certain cases for two successive years. Regulations have also been put into operation so as to prevent the disease being transported from place to place in bushes or cuttings. The success which has attended these efforts to combat the fungus has been, it would appear, very uncertain owing, largely, to the incompleteness of our knowledge of the life-history and mode of development of the fungus during the resting period of the disease. The result has been that the growing of gooseberries has, in general, decreased and in some districts has probably had to be given up.

Control Measures:

(1) All severely affected bushes in the plantation or any wild bushes in the vicinity should be dug up and burnt. (2) Less severely affected

bushes should be pruned after the leaves have fallen in the autumn and the diseased shoots collected and burnt. (3) The pruned bushes, as well as the soil below, should then be sprayed thoroughly with liver of sulphur (3 per cent. solution), formalin (1 in 40), or some similar fungicide. (4) In spring, immediately before flowering, spray with Burgundy mixture, which, according to Dutch experience, protects the young fructifications and makes possible the production of normally developed, ripe berries (Maarschalk, 1923).

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Sphærotheca Fragariæ (Harz) Arth. is a form of mildew which appears on the strawberry (*Fragaria* cult.) and is closely allied to the hop mildew with which it is generally identified. In its conidial stage (*Oidium Fragariæ* Harz) the fungus attacks the leaves of the strawberry plant and causes them to curl at the margins so that the under surface is turned upward and the plants appear to be dried up. The white mycelial felt is limited almost entirely to the under surface of the leaf. In severe outbreaks the fungus attacks the fruit in all stages of development, the green berries shrivel and have the appearance of having been dusted with flour. The fungus does not, apparently, go beyond the conidial stage, but in America, later in the summer, perithecia have been found (Salmon, 1901). The disease was first mentioned in 1854 in England;

in 1886 it was reported from America, in 1887 from Germany, and in 1910 in Sweden.

Powdery Mildew of Hop

Sphærotheca Humuli (DC.) Burr.

This fungus produces white powdery patches on the leaves of the hop (*Humulus Lupulus*). The patches are small at first, but they gradually increase until the whole surface of the leaf is covered with a white, flour-like dust. From the superficial mycelium, haustoria pass downwards into the epidermis whilst numerous erect conidiophores arise externally. The chains of conidia and the detached conidia impart a floury appearance to any parts of the plant that are suffering from the disease. The hop cones are also attacked in a similar manner and the yield may be seriously affected.

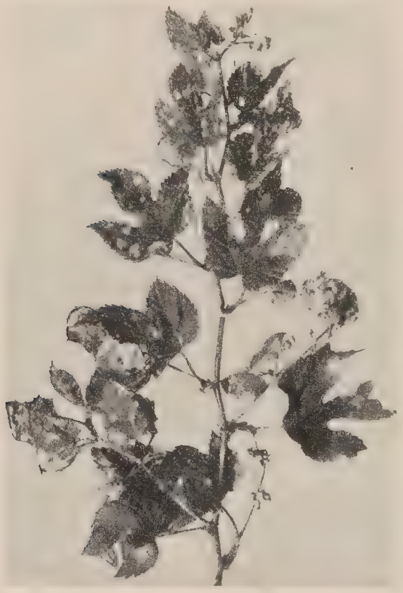

Fig. 238.—Hop Powdery Mildew. (After F. M. Blodgett.)

After a short time, small, black dots appear in the grey coating of mycelium and it is from them that the perithecia, which are designed for the over-wintering of the fungus, arise. In spring and in damp weather the asci in the perithecia are liberated, and the ascospores are then ready to germinate and cause fresh infection.

The older varieties of European hops do not show any appreciable differences in their resistance to the fungus ; in a bad mildew year all suffer severely and the weight and quality of the crop are very much reduced. The raising of new varieties resistant to the fungus appears to offer some hope of controlling the disease. Thus in the years 1914–20 experiments were carried out in England (Wye College) with plants raised from seeds obtained from Italy. In the greenhouse twenty-seven forms proved to be immune to mildew and seven forms were " commercially resistant." The majority of the forms which had proved to be immune retained this property when grown out-of-doors and appeared to be uninfluenced by external conditions (weather, proximity of diseased plants, etc.). The property of resistance seems, therefore, to be constitutional. Unfortunately these resistant forms are, in other respects (time of flowering and ripening, size of cone, richness in resins, aroma), not as good as the older varieties, so that their commercial value is low. It is hoped, however, by cross-breeding with the old and new varieties, to obtain plants suitable for commercial cultivation.

In England, dusting with flowers of sulphur is regarded as the best means of controlling the disease. Other sulphur preparations such as liver of sulphur, lime sulphur, and Solbar have also been recommended as suitable fungicides.

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The forms of mildew on a number of other plants, as for example species of the genera *Alchemilla*, *Rubus*, *Potentilla*, *Geranium*, etc., have been regarded by some mycologists as the same species as the hop mildew, but it is not yet possible to say whether this view is correct. According to recent investigations the forms of mildew on species of *Alchemilla* form a separate species, **Sphærotheca Alchemillæ** Stein., within which are specialised forms of the various groups of *Alchemilla* such as *Vulgares*, *Alpinæ*, *Pubescentes*, etc. (Steiner, 1908).

Microsphæra

The perithecia contain several asci and each ascus eight spores. The appendages are straight, branched several times dichotomously or trichotomously at the apex.

Microsphæra Betæ Vanha was noticed in the years 1899–1902 on sugar beets in Moravia. A white coating, which increases until the leaf has a floury appearance, is found on both surfaces. The leaf loses its gloss and becomes of a dull, yellowish-grey colour. Later the perithecia appear as brownish-black dots (Vanha, 1902).

Oak Mildew

Microsphæra quercina (Schw.) Burr.

This fungus produces white, cobweb-like patches of varying size upon the upper surface of the leaves of the oak. The patches are isolated at first, but they soon spread and finally cover the whole of the upper surface with a white, powdery coating which adheres firmly to the leaf and cannot be wiped off.

In many cases the outbreaks of the disease occur at two different periods; the first, which is slight and less noticeable, is in spring (May) shortly after the leaves appear on the one-year-old shoots, and the second in summer or autumn (August and September) on the young, tender leaves of the summer shoots. This latter outbreak appears mainly on young trees and on stool-shoots, especially in places where the ground has been cleared of trees and replanted. A severe outbreak can be recognized from a distance, as the leaves have lost their green colour almost entirely and the trees look as though they had been sprayed with lime-wash. The leaves finally become brown and shrivelled and fall prematurely. The tips of the shoots do not ripen but wither and die back during the winter. As the fungus has a preference for one- to six-year-old plants it is especially dangerous in nurseries. The white coating consists of conidiophores and conidia produced by them; the conidia are able to germinate at once, and so the disease is quickly spread.

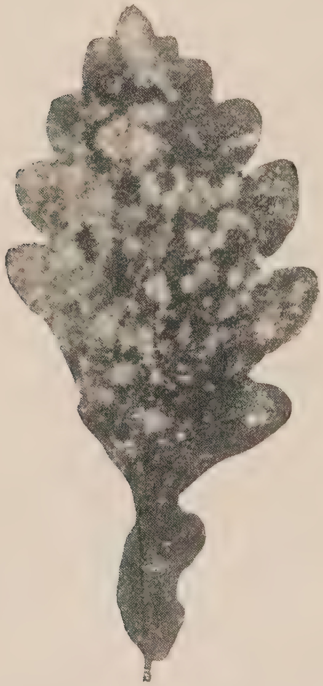

FIG. 239.—Oak Mildew. Oidium stage. (Orig.)

Efforts to discover the perithecial stage were fruitless until 1911, when it was observed in France on small, sickly-looking, but still living leaves of *Quercus sessiliflora*. The perithecia appeared as small, black dots in the white mycelium. The appendages were long and much branched at the tips, producing a coral-like appearance. On account of the structure of the perithecia the fungus is placed in the genus *Microsphæra*. The perithecial stage was later discovered in other countries—1919 in Italy (Bologna, Sicily), 1921 in Germany (Hildesheim), 1920–22 in

Central and Northern Russia. The perithecia, as a rule, only appear on older leaves, but they are found there in large numbers.

The fungus was first observed in Europe (France) in 1907; in the following year patches of mildew appeared on oak leaves already in May. The disease spread with great rapidity, and by the autumn of the same year it was known in almost all parts of France. The fungus quickly spread to other countries and was found in 1908 in Spain, Portugal, Algiers, Italy, Switzerland, Austria, Hungary, Germany, Holland, Belgium, Luxemburg, England, Sweden, and Denmark, but only in the conidial stage. How the mildew came to appear so suddenly throughout Europe is not known; possibly it migrated from North America or it may have previously existed in Europe in a harmless and unnoticed form which then, through unknown circumstances, became epidemic.

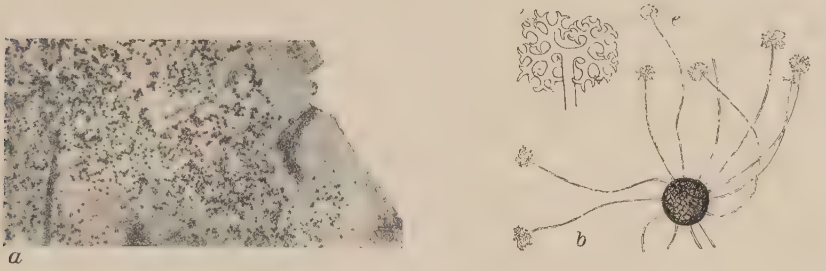

FIG. 240.—Oak Mildew. Perithecial stage.

a, portion of leaf; *b*, perithecium with appendages. (*a*, after A. Buchheim; *b*, after E. Fischer.)

In view of the rare appearance of the perithecial stage in Europe the question of the over-wintering of the fungus was not decided until it was observed, in Germany (Laubert, 1910; Neger, 1911) and in Italy (Peglion, 1911), that the mycelium can hibernate in the winter buds of the oak. The mycelium passes the winter between the scales of the buds and in spring puts out conidiophores from which conidia arise and infect the young tender leaves.

The fungus shows a preference for European species of the oak such as *Quercus robur*, *Qu. sessiliflora*, *Qu. pubescens*, *Qu. Cerris*, *Qu. tozza*, *Qu. crispula*, etc.; the American varieties, *Qu. rubra*, *Qu. coccinea*, *Qu. palustris*, etc., are less susceptible. In rare cases the fungus attacks stool-shoots of beech (*Fagus silvatica*) and chestnut (*Castanea vesca*).

Spraying with lime sulphur (1 : 20) or with brine—10 lbs. salt to 5 gallons for young leaves and 10 lbs. to 3 gallons for older leaves—is recommended, or the trees can be dusted with sulphur, once when the leaves have appeared and a second time after the development of the summer shoots.

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Microsphæra Grossulariæ Lév., European Gooseberry Mildew, covers the leaves and parts of the berries with a very delicate, greyish-white network in which the perithecia are embedded as small, black dots. The perithecia contain 4–8 asci with 4–5 spores in each ascus. The appendages are dichotomously forked at the tip. This disease does not generally cause much damage.

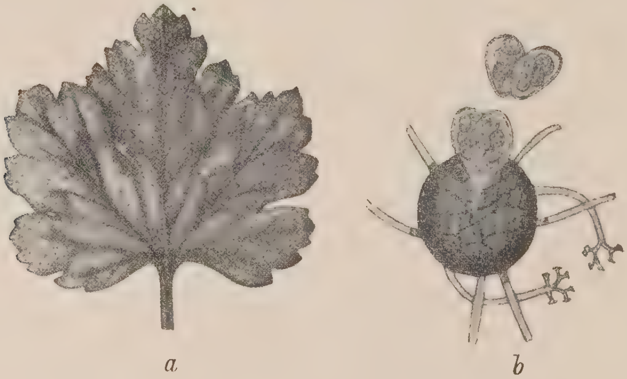


FIG. 241.—*Microsphæra Grossulariæ.*
a, diseased leaf; *b*, perithecium with emerging asci. (Orig.)

M. ferruginea Eriks. sometimes appears on *Verbena hybrida* growing in the open. The fungus is mainly found on the under surface of the leaf, which becomes of a pale rust-red colour with here and there patches of mildew of the same colour, which finally cover the whole leaf. In late summer, small, brownish-black perithecia are found sparingly in the mycelium on the upper surface of the leaf, and their structure shows that the fungus belongs to the genus *Microsphæra*. This fungus was found in a Stockholm garden in 1882–84 and was so severe that in spite of heavy dustings of sulphur the growing of the verbena had to be abandoned (Eriksson, 1885).

Oidium Euonymi-japonici (Arc.) Sacc. The evergreen shrub, *Euonymus japonicus*, which is largely used for decorative purposes, is attacked by this fungus. Small, round, isolated, later coalesing, patches of white to greyish-yellow mycelium are formed on the leaves. In the middle of these patches the hyphæ are closely interwoven, giving a scurf-like appearance to the mycelium. In severe attacks of the disease the leaves of the plant fall and its value for decorative purposes is lost.

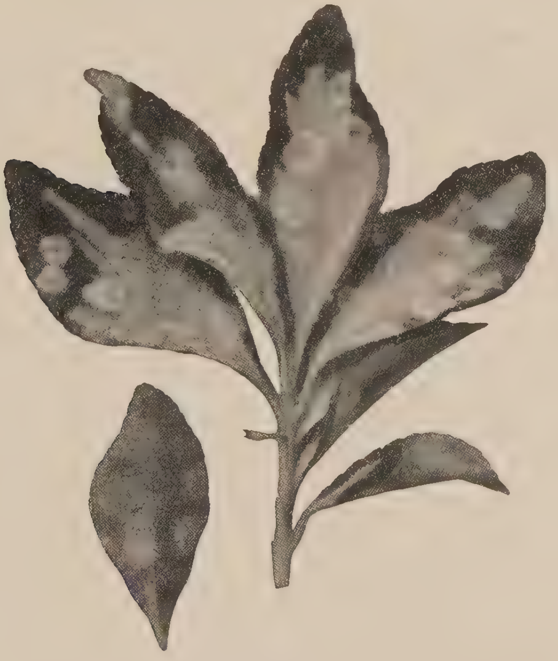


FIG. 242.—*Oidium Euonymi-japonici.* (Orig.)

By artificial inoculation the fungus has been transmitted to a number of varieties of the host plant, *e.g.* var. *aureus*, var. *ovatus aureus*, var. *microphyllus*, var. *albo-marginatus*, and var. *President Gunter* as well as to *Euonymus radicans* var. *microphyllus* and var. *Silver Gem*; on the other hand, *Euonymus europæus*, *E. nanus*, and *E. chinensis* proved to be immune. Uninjured leaves can only be infected when young; for infection to take place on an old leaf it is necessary that a wound should exist; such would, however, be very likely to occur on plants in the open through injuries by insects or other causes (Salmon, 1905, 1906).

The perithecial stage of the fungus has not been observed and the fungus lives from year to year in the mycelial stage. It has been found that certain coarse felts of mycelium, of an almost crust-like nature, which are found on the leaves can hibernate and develop further in the following spring. Conidiophores and conidia arise from this mycelium and lead to a new outbreak of disease (Salmon, 1905; Foëx, 1910).

This fungus is a native of Japan and was first observed in Italy, 1899–1905; England (Bexhill), 1903; Austria and Southern Tyrol, 1904; France, 1904; Sweden, 1906.

To control the disease it is recommended to collect and burn, in the autumn, all leaves covered with mildew or, if such a course is impracticable, the infected plants should be dusted with sulphur or sprayed with a solution of liver of sulphur.

Oidium Chrysanthemi Rab. appears as a white, floury coating on the leaves of *Chrysanthemum indicum* in the greenhouse during the months of October and November. The floury appearance is due to the conidiophores and conidia of the fungus. The perithecial stage of the fungus has not yet been observed. Diseased plants are considerably retarded in their general development and in the production of flowers. The fungus was first reported in 1853 from Germany (Dresden); it has also appeared regularly, almost annually, on chrysanthemum grown near to Stockholm (Eriksson, 1885; Laubert, 1909).

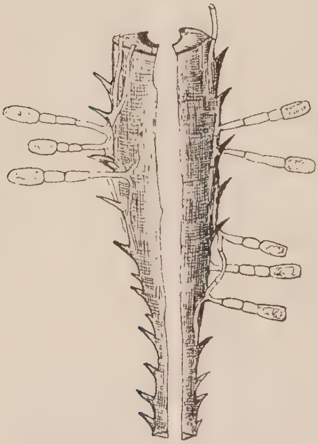


Fig. 243.—*Oidium ericinum.* (Orig.)

O. ericinum Eriks. attacks the leaves and stems of a number of species of *Erica*, *e.g.* *Erica gracilis* and *E. persoluta*, in summer (July) when the plants are out-of-doors, sometimes at other periods of the year as well. The mycelial coating is very thin and the conidiophores stand widely apart. The older twigs have a greyish lustre; in the case of severe outbreaks the individual twigs become almost white. The needle-shaped leaves turn brown from the base upwards. The fungus causes the leaves to fall and often kills the plant. Perithecia have not been found. This form of mildew appeared at the beginning of the eighties of last century near Stockholm (Eriksson, 1885), in 1909 in Bernau (Brick, 1910), in 1910 near Dresden (Naumann, 1912), and in 1911 in Vienna (Köck, 1911), the outbreaks varying in intensity. **O. Hyssopi** Eriks. appeared near to Stockholm in the summer of 1882 on *Hyssopus officinalis* growing in the open. The attack was severe and began early in July. Towards the end of the month the diseased plants were completely enveloped in a white coating ; the number of plants which suffered was considerable, many dying completely. Perithecia were not found. The disease was not noticed in 1881 nor in 1883, although a considerable amount of hyssop was then being grown, particularly in the latter year (Eriksson, 1885).

CHAPTER XI

PERISOPORIACEÆ

Root Rot

Thielavia basicola (B. & Br.) Zopf.

This fungus appears on the roots of tobacco, lupine, violet, pea, turnip, begonia, etc., and causes them to shrivel up and become soft, the leaves and stem cease to grow, and the flowers do not develop. On older plants in the open the main root is more or less rotten and breaks off if drawn from the ground. The lateral roots are stunted and bunched together at the neck of the root.

The fungus develops unicellular endospores which are able to germinate at once when ripe and also multicellular, thick-walled chlamydospores which, after hibernation, germinate in the following spring. A third method of reproduction, when conditions are favourable, is by ascospores which arise from black perithecia. Spore formation takes place not only on the surface of the roots but also within the root cells.

The soil is infected by the chlamydospores and ascospores which over-winter there and germinate in the spring. When healthy seedlings are planted in such infected soil they grow badly or die. On the other hand, seedlings which are only slightly attacked by the disease may give relatively good crops if planted in disinfected soil ; for the purpose of disinfection a solution of formalin (1 per cent.) is recommended.

The disease was first noticed in 1850 in England on peas, and in 1876 in Germany on lupines and other plants. In 1891 it appeared on scented violets in America and in 1896 on tobacco plants. Since the last date the fungus has been a serious pest in tobacco cultivation, not only in America (from Ohio eastwards) but also in Europe (from England to Italy). In recent years it has appeared in Norway (Indre Sogn) and caused much loss.

Control Measures :

(1) Plants susceptible to the disease should not be grown in infected soil. (2) The frames in which the seedlings are raised should be well ventilated, and in field cultivation the soil should be efficiently drained.

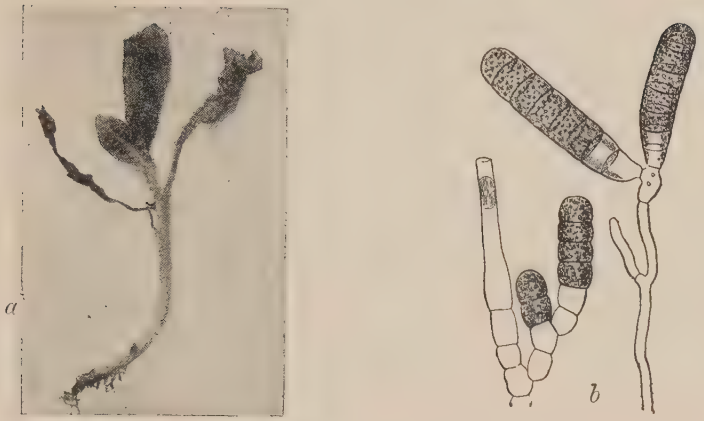


Fig. 244.—Root Rot.

a, diseased tobacco plant; *b*, mycelium with chlamydospores on lupine. (*a*, Orig.; *b*, after W. Zopf.)

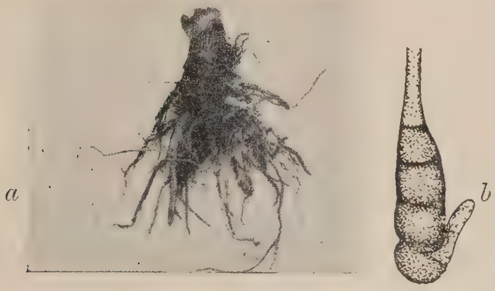
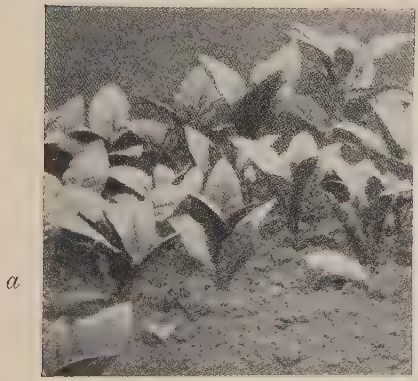


Fig. 245.—Root Rot.

a, diseased root of tobacco plant; *b*, chlamydospore, germinating. (After G. P. Clinton.)



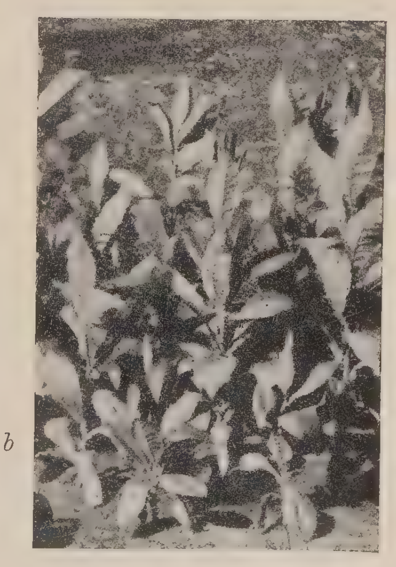


Fig. 246.

a, tobacco plants, originally healthy, attacked by disease through being planted in infected soil; *b*, tobacco plants originally diseased but recovered on planting in fresh, uninfected soil. (After G. P. Clinton.)

(3) Diseased seedlings should not be planted out. (4) The soil in the frames should be disinfected; 1 gallon of 40 per cent. formalin to 100 gallons of water. From 5 to 6 gallons of this solution should be used to water a square yard of soil.

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CHAPTER XII

HYPOCREALES

THE perithecia are globose or ellipsoid with an apical opening. The perithecia are soft and generally of a bright colour, never hard and black.

Snow Mould Disease of Cereals. Seedling Blight.

Calonectria graminicola (B. & Br.) Wr.

Fusarium nivale Ces.

In northern countries when the snow melts in spring, especially after a winter with a heavy snow-fall and not very severe frost, a white or reddish-grey coating appears on the shoots and leaves of autumn-sown cereals as well as on meadows and pastures. The mycelium is often very scanty at first, but under favourable conditions (suitable temperature, atmospheric moisture, etc.) it quickly develops and finally produces a thick coating which covers the plant as with cotton-wool. If, then, a drying wind is not obtained the plants begin to rot and bare patches of varying size appear in the field. When the snow melts slowly the havoc caused by the disease is greater.

The mould consists of interwoven, sinuous, branched hyphæ in which a large number of gelatinous, reddish clumps, composed of innumerable conidia, occur. When the conditions are favourable the conidia germinate and spread the disease. In this stage of its development the fungus is known as *Fusarium nivale* Ces. Other species of *Fusarium* when in association are also the cause of snow mould on wintered cereal plants in the spring, especially *F. rubiginosum* App. & Wr.; this species alone can give rise to the disease.

Shortly after the mould-covered leaves have wilted, the fungus enters on another stage of development in which small, black, spherical perithecia, each with a round apical pore, are found in groups along the veins of the leaf. This stage is named *Calonectria graminicola* (B. & Br.) Wr. When the perithecia are ripe the spores which they produce serve to infect other parts of the cereal plant. In wet summers the fungus gives rise to a disease which is similar to Foot Rot (*Leptosphæria herpotrichoides* and *Ophiobolus herpotrichus*) and known as "straw Fusarium"; this disease causes the stem to break off at, or near, the ground. The fungus ultimately appears in the ears, especially in the case of rye, and is often associated with other species of *Fusarium.* When infection occurs during the development of the seed, the grains are much shrivelled, but if infection takes place after the seed has fully developed—shortly before or during harvest—the shrivelling is only slight and at the base of the grain.

From the mycelium there are developed, particularly when there is lack of nourishment, intercalary chlamydospores, which are usually one-celled, ovate, and separate, as well as sclerotia-like structures which over-winter on fallen leaves and in spring give rise to fresh outbreaks of disease.

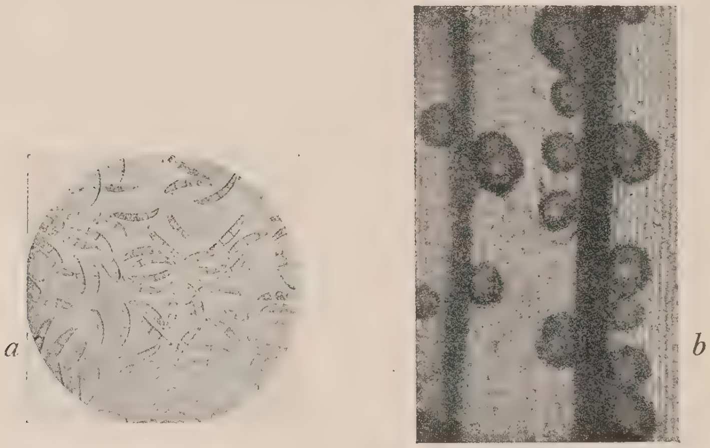

Fig. 247.

a, Fusarium nivale. Conidia. (After E. Schaffnit.) *b, Calonectria graminicola* on a dead leaf of rye. (After L. Hiltner and G. Ihssen.)

Seedlings from badly-infected grains develop abnormally and instead of growing up through the soil in the normal way they bend in the ground and grow sideways. The disease is usually carried in the seed, but there is also infection from the soil.

This fungus was described more than 100 years ago under the name of *Lanosa nivalis* (Fries, 1825); rye is the most susceptible cereal, and then wheat. Observations made in Prussia from 1912 to 1916 showed that 11–25 per cent. of the rye-fields were severely affected. The fungus can also pass to other plants such as clover, peas, etc., and take part in producing a disease similar to Foot Rot.

Control Measures :

(1) To hasten the melting of the snow, any heaps that are present on cornfields or meadows should be scattered. (2) Surface channels should be cut to allow the water from the snow to drain away quickly. (3) The field should be lightly harrowed early in spring. (4) A top dressing of kainit will hasten the melting of the snow, as a rule, when the temperature is above zero and it also manures the crop. (5) Good plump seed with a high 1.000 grain weight and germinating power of not less than 95 per cent. should be chosen for sowing. (6) For drilling 90–100 lbs. of seed per acre and for broadcasting 130–140 lbs. should be used in the case of rye. (7) Rye should not be sown too early, otherwise the growth may be too luxuriant. (8) The seed should be dressed with a disinfectant if there is any suspicion that it is infected.

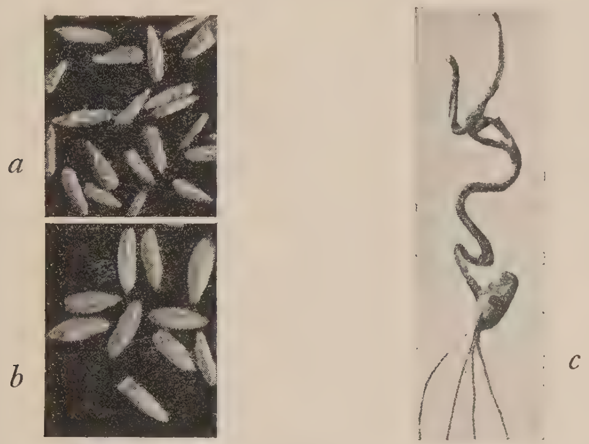

FIG. 248.

a, primary (during the development of the grain) infection of rye grains by *Fusarium nivale* ; *b*, secondary (shortly before harvest) infection of grains. (After E. Schaffnit.) *c*, diseased grain of rye, germinated but unable to reach surface of soil. (After L. Hiltner and G. Ihssen.)

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Gibberella saubinetii (Mont.) Sacc., Wheat Scab, occurs on the grains and glumes of wheat, maize, barley, rye, etc. The patches of mycelium are reddish and effused, and from them spindle-shaped, curved, six-celled conidia of a reddish colour are developed. In this conidial stage, *Fusarium rostratum* Ap. & Wr. (also named *F. roseum* Link. and *F. graminearum* Schwabe) has been known for a long time as a disease of European cereals, and it has latterly caused heavy losses in some of the central states of North America. When the conditions are favourable, the perithecial stage develops in the form of small, shining dots on the grain. The ascospores are ellipsoidal and divided by cross walls into four cells. The fungus is transmitted to the next year's crop by infected seed and infected soil (Atanasoff, 1920 ; Dickson and Johann, 1920 ; Dickson, 1923 ; McInnes and Fogelmann, 1923).

Diplodia Zeæ (Schw.) Lév. is the cause of a " dry rot " in maize grains in America (Nebraska) (Heald, Wilcox, and Pool, 1909 ; Durrell, 1925 ; Reddy, Holbert and Erwin, 1926).

Bullrush Fungus of Grasses

Epichloë typhina Tul.

This fungus is found mainly on *Phleum pratense* and *Dactylis glomerata*, but many other species of the genera *Poa*, *Agrostis*, *Calamagrostis*, *Milium*, *Holcus*, *Bromus*, *Schedonorus*, *Anthoxanthum*, etc., are also attacked by it. The fungus appears at first as a greyish-white coating, which later becomes golden-yellow to brown, on the sheaths of the upper leaves. The appearance of the infected part bears a resemblance to the female inflorescence of the bullrush (*Typha*), and this has given the species-name to the fungus. As a consequence of the disease the growth of the shoot ceases and no inflorescence is formed. The greyish-white coating (stroma) is composed of closely-crowded hyphæ which extend radially outwards and produce unicellular conidia at their tips. As the stroma changes in colour from white to deep orange the hyphæ are replaced by others which develop into asci, each of which contains eight long, narrow, jointed spores. It is thought that the conidia spread the disease from plant to plant in the summer, whilst the ascospores keep the fungus alive from year to year.

To test this view, inoculation experiments were carried out under glass in Sweden in 1902 with a number of grasses susceptible to the disease. Very small pieces of infected timothy grass, on which white conidia were present, were attached to the young leaf sheaths of grass seedlings : *Dactylis glomerata* (6 plants), *Holcus lanatus* (6), *Poa nemoralis*

(5), *P. pratensis* (6), *Phleum Böhmeri* (3), *Phl. pratense* (3). After thirty-nine days there was no trace of disease to be seen in any case; possibly a longer incubation period, say of a year, is necessary. It seems as though this fungus, when it has once entered the host plant, can live in the main axis of perennial grasses and may possibly be disseminated in the seed.

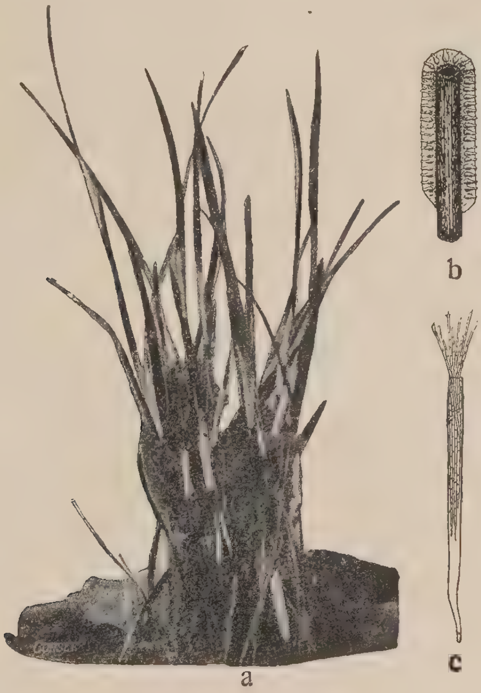

Fig. 249.—*Epichloē typhina.*
a, diseased stems of timothy grass; *b*, longitudinal section of stroma with numerous flask-shaped perithecia; *c*, ascus with spores. (*a*, after K. v. Tubeuf; *b* and *c*, after E. Rostrup.)

In some years, fields of timothy suffer severely from this disease, one of these cases being observed in Sweden in 1902 when the crop was reduced by two-thirds; cocksfoot and other grasses in the district were free of the disease or only slightly affected. The following year (1903) no signs of the disease were observed on the timothy grass. In Hungary the fungus has been found on *Agropyrum repens* (Pater, 1913).

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Ergot

Claviceps purpurea (Fr.) Tul.

Sphacelia segetum Lév.—*Sclerotium Clavus* DC.

Ergot is the name given to the large, hard, purplish-black bodies which appear on the ears and panicles of a number of cereals and grasses in place of the ordinary grain or seed. These ergots are white within, and several may be present in the same ear, especially in the case of rye, which led to the old name of the disease being *Secale cornutum*.

The ergot is a sclerotium designed for the hibernation of the fungus, and at harvest the ergots separate from the ear and fall to the ground or else remain attached and become mixed with the threshed grain. When the ergots lie in the soil over the winter they germinate in spring, and from each ergot there grow out a number of stromata, often ten or more, each with a thin, reddish-yellow stalk and a round, dark red head. The stalk is sufficiently long to allow the head to appear above the

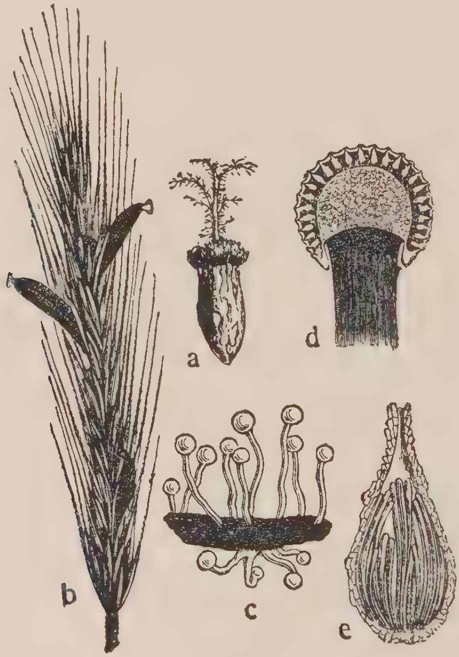

FIG. 250.—Ergot on Rye.

a, developing ergot ; *b*, ear of rye with 3 ergots ; *c*, over-wintered ergot with 13 stromata ; *d*, head of stroma in section, showing perithecia ; *e*, perithecium with asci. (After E. Rostrup.)

ground. All round the surface of the head are embedded flask-shaped perithecia which are filled with long asci; in each ascus there are eight slender ascospores. These spores are freed at the time when the cereal or grass in question is in flower and are distributed by the wind; they enter the open flower, adhere to the stigma, germinate, and grow down into the young ovary. A single ergot is able to infect the inflorescences of hundreds of rye plants.

The infected ovaries do not develop normally, and from the surface a pale yellow, sweet, sticky fluid, which resembles the "honey dew" produced by aphides, is secreted and is often given the same name. This fluid contains an enormous number of small conidia which form the summer stage of the fungus. For some time this stage of development was regarded as a special form of fungus and was named *Sphacelia segetum* Lév. By means of the conidia the fungus is carried to healthy inflorescences on the same or other ears. The distribution takes place either through the exuded "honey dew" being carried from ear to ear by contact as the ears move in the wind, or by the agency of insects. The infected ovaries finally assume the form of an ergot. Infection can also take place in the following year by means of hibernated conidia, if such conidia are carried to the open flowers.

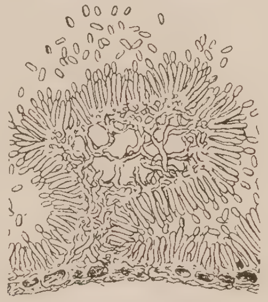

Fig. 251. *Sphacelia segetum.* (After E. Prillieux.)

Rye is the most susceptible of the cereals; sometimes 10 per cent. of the ears are found to contain ergots. Diseased ears ripen at a later date and yield a larger number of shrivelled or blind grains than do the healthy ears (Seymour and McFarland, 1921). Ergot only appears sporadically on barley and wheat, which is probably due to the differences which exist in the flowering stage of these two cereals. The flowers of rye remain open for hours to infection by the visits of insects or by wind, whereas wheat and barley keep their glumes closed and the ovary protected. Ergot has been found on oats in Algeria and North America.

Several biologically specialized forms of this species of fungus have been distinguished up to the present: (1) f. sp. Secalis on rye, more rarely on barley, wheat, and oats, also on *Anthoxanthum odoratum, Hierochloa borealis, Arrhenatherum elatius, Dactylis glomerata, Hordeum murinum, Festuca pratensis, Phalaris arundinacea, Briza media,* and *Calamagrostis arundinacea;* the fungus can likewise be easily transmitted to *Poa pratensis, P. cæsia, P. sudetica, P. hybrida,* and *P. compressa.* (2) f. sp. Lolii on *Lolium perenne, L. italicum, L. temulentum, L. rigidum,* and *Bromus erectus.* (3) f. sp. Poæ annuæ on *Poa annua;*

and (4) f. sp. Brachypodii on *Brachypodium silvaticum* and *Milium effusum*. The latter form is peculiar in that it develops conidia in considerable numbers, but only rudimentary sclerotia, on varieties of *Milium*, which flower early, whereas on species of *Brachypodium*, which flower later in summer, it produces conidia and fully developed sclerotia.

Ergot is poisonous and bread made from rye which contains much ergot produces a disease (Ergotism) which is characterized by attacks of fainting, convulsions, and paralysis. In earlier days, epidemics of this disease occurred in which only 5 per cent. of the people who suffered from it survived; cattle and poultry are also subject to ergot poisoning. When ergoted corn is stored its poisonous properties decrease and finally disappear, but as a safeguard ergots should always be removed from grain before it is fed to cattle or ground into flour.

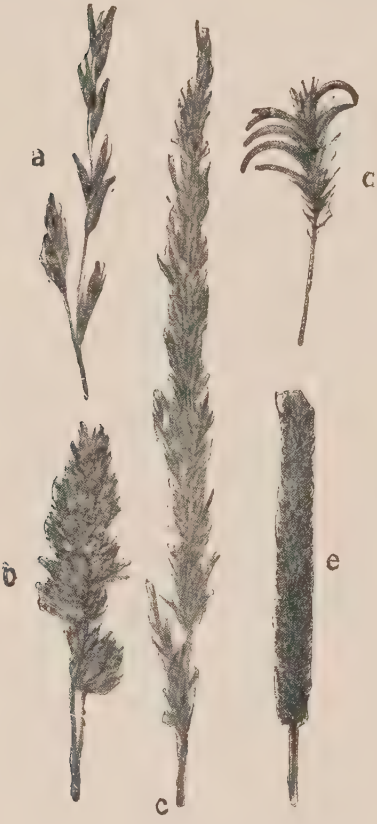

Fig. 252.—Ergot on Species of Grasses. *a*, *Festuca arundinacea*; *b*, *Dactylis glomerata*; *c*, *Calamagrostis arundinacea*; *d*, *Triticum desertorum*; *e*, *Phleum pratense*. (Orig.)

As the stock of ergot, which is of value for medicinal purposes, was much reduced after the World War owing to the falling off in supplies from Russia and Galicia, steps have been taken to produce ergot artificially (Hecke, 1921; Falck, 1922).

Control Measures:

(1) Any ergots present in corn or grass seeds should be removed by appropriate sifting and winnowing so as to prevent infection of the next crop or injury being caused if the corn is used for food. (2) Cereal grains or grass seed, if contaminated with pieces of ergot, should be treated by stirring in a vat with a solution of potassium chloride (32 per cent.) for quarter of an hour. The pieces of ergot should then be skimmed off and the grain or seed washed with water and spread out to dry. (3) As the little heads, which are produced when an ergot germinates, have only relatively short stalks, deep ploughing of the stubble in autumn will cover the ergots to a depth which will prevent the heads from

reaching the surface and so remove this source of infection. (4) As the ears of corn can only be infected at the time of flowering, this period should be rendered as short as possible by attention to the manuring of the crop and by sowing the seed at a uniform depth. It is also desirable not to grow early and late sorts of the same kind of cereal near to each other. (5) If there be reason to fear that wild grasses growing at the sides of the field, etc., may develop ergot they should be mown down at the time of flowering and so prevent infection of the cereal crop.

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Very closely related to the fungus just described are **Claviceps Wilsoni** Cooke (?=*Cl. purpurea* f. sp. *Glyceriæ* Stäg.) on *Glyceria fluitans* and **Cl. microcephala** Tul. on *Phragmites communis*, *Molinia cærulea*, *Nardus stricta*, and *Aira cæspitosa* (Stäger, 1903).

Apple and Pear Canker

Nectria galligena Bres.

Fusarium Willkommi Lindau

This fungus attacks the branches of the apple tree preferably, but it is also found on pears and other trees. In one form of the disease—open canker—small or large wounds bounded by a raised layer of cracked bark are found on the branches. These wounds enlarge and may ultimately girdle the branch. In the other form—closed canker—swellings of various sizes and shapes occur, chiefly in the angles of the branches. The wood grows irregularly and the fruit which is formed is dwarfed and drops prematurely. The cankered, stunted shoots and branches gradually die and the tree finally perishes.

In the open wounds there are considerable accumulations of mycelium usually with stromata of two kinds, one greyish-white, which bears conidia, *Fusarium Willkommi* Lindau, the other with crowded, red perithecia, *Nectria galligena* Bres. Branches of the mycelium spread into the vessels and wood cells and from these points into the starch-containing cells.

It is generally assumed that the fungus is not able to penetrate the undamaged bark but finds entrance through wounds caused by frost, hail, insect injury, or where branches have broken off. Once the mycelium has entered the branch it spreads rapidly in the bark during early spring or late autumn; in summer growth is at a standstill. Owing to this periodic growth the wounds can, under favourable conditions, be partially covered over by the formation of callus round the edges. When, however, the mycelium enters upon another period of growth the edges of the wounds are again destroyed, so that gradually the cankers become larger and deeper. The fungus requires a considerable degree of moisture for its vegetative growth. The perithecia arise, either singly or in groups, on the surface of a red stroma. They are at first of a cinnabar-red colour, later scarlet or nearly black, ovoid in shape, with a dark, hyaline, almost black, beak or a more or less well-marked ostiolum. Spherical, conical, or bristle-like excrescences occur on the upper surface of the perithecia. The spores are hyaline, ellipsoid to ovoid in shape, two-celled; the paraphyses are long and jointed.

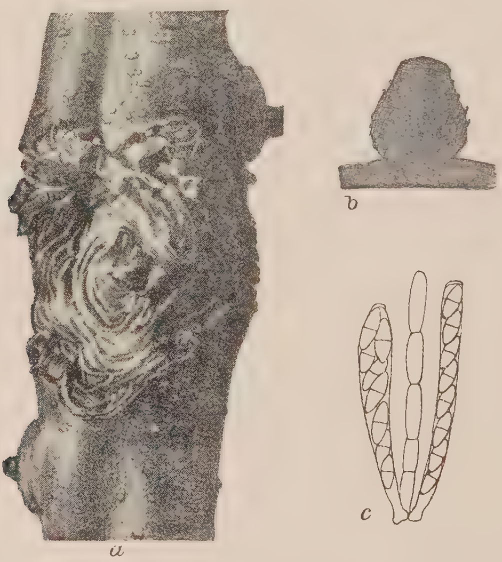

Fig. 253.—Apple Canker.

a, cankers on stem; *b*, perithecium; *c*, two asci and a paraphysis. (*a*, after R. Göthe; *b* and *c*, after J. Weese.)

It has been noticed that in some instances the conidial stage of the fungus, *Fusarium Willkommi*, alone has been the cause of injurious growths on the previous year's shoots of the apple tree and on the young fruits of apple and pear. A case of this kind occurred in a nursery in Sweden (Kungsbacka) in 1913, where at the end of May the previous year's shoots on apple trees of several varieties were swollen at the nodes and the swellings covered with greyish-white stromata arranged in groups. At a number of the nodes the buds, with their leaves, had already fallen. During the previous winter all the varieties of apple trees in the nursery were apparently healthy, also in March when they were pruned. In the fourth week of April there was frost at night. This was followed by warmer weather and the leaves developed further; then came another spell of cold weather but no night frosts. The conidia were slightly bent, with 3–5 septa.

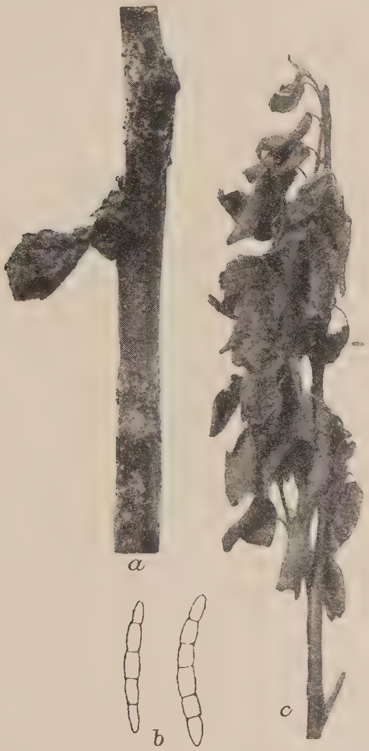

Fig. 254.—Apple Canker. Fusarium stage (*Fusarium Willkommi*) on one-year-old apple twigs. *a*, end of May; *b*, Fusarium spores; *c*, beginning of July. (Orig.)

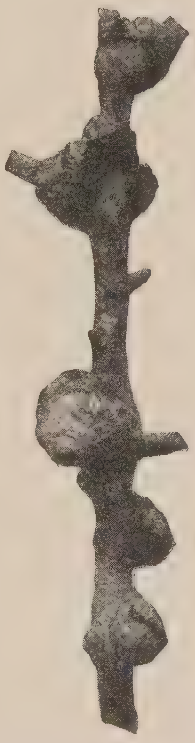

Fig. 255.—Apple Canker. Closed cankers. (Orig.)

Similar *Fusarium* attacks on ripening fruits of pear and apple were observed in Denmark in 1919 and 1920. Sunken, sharply-defined, brown patches of decay occurred on the fruits, and in these patches were found the greyish-white conidial stromata of *Fusarium Willkommi*. By artificial inoculation with conidial material taken from diseased pears it was possible to infect healthy fruits of pear and apple trees, and also the internodes of young shoots of birch and apple. With material taken from the lesions on the wood it was possible to again produce the *Fusarium* stromata on fruits of apple and pear, provided that the epidermal tissues were injured and so allowed the fungus to enter.

It is a question whether the sudden appearance of such an independent and abnormally active conidial stage is to be regarded as due to biological changes in the fungus, or whether it is a purely accidental phenomenon caused by external conditions (weather, soil).

" Closed " canker differs from the " open " form just described in having knotted outgrowths and also by the cankers being completely covered over. This form of canker has been known from earliest times in all European countries, and latterly it has been observed in America (Zeller and Owens, 1921).

Nectria galligena, sometimes with and sometimes without the formation of cankers, has also been found on *Salix purpurea*, *S. Caprœa*. *Populus nigra*, *Rhamnus Frangula*, *Corylus Avellana*, and species of *Quercus*, *Fraxinus*, and *Ribes*.

Some scientific investigators, as well as some practical gardeners, are of the opinion that a fungus is not the primary cause of canker and only plays a secondary part in the process of destruction. This opinion is based mainly on the appearance of canker where no species of *Nectria* is to be found and to the equally common observation that the *Nectria* fungus occurs without the formation of cankers. The origin of the cankers and galls is considered to be due to an inherited tendency of the individual to hypertrophy of the wood cells in consequence of certain injuries and in unfavourable soil conditions (Sorauer, 1886).

In 1903, Brezezinski published the results of his investigations, which had been carried out for many years, in connection with canker on apple, pear, and hazel-nut trees, and expressed the opinion that the disease is not due to a *Nectria* fungus but is a consequence of the bacterial invasion of the branches (see section on Bacterial Diseases of Fruit Trees, etc., p. 31).

Control Measures :

(1) The open and closed cankers should be cut out in winter and the wounds painted with warm coal tar. (2) Varieties susceptible to canker should not be planted ; local experience in this respect should have consideration.

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Nectria coccinea (Pers.) Fr., usually called *Nectria ditissima* Tul. formerly, agrees generally with the above fungus. It causes closed or open cankers on a number of varieties of trees such as horse chestnut, beech, oak, maple, elm, acacia, etc. These two species of *Nectria* can only be distinguished by their choice of host, the structure of the perithecia, and the dimensions of the ascospores. In the case of *N. coccinea* the perithecia are ovoid or lemon-shaped without a marked neck and with an apical ostiole embedded in a pit-like depression of the perithecial stroma. The length of the ascospores is 12–13 μ according to Dutch measurements (Westerdijk and van Luijk, 1924) or 8–14 μ according to American (Zeller, 1922), whilst for *Nectria galligena* they are given as 17–18 μ or 14–22 μ respectively.

The two species do not, however, appear to be sharply separated in their biological characters. *N. galligena*, although it attacks pomaceous trees, is also found on *Salix* and *Fagus*. *N. coccinea* appears on a large number of foliage trees, apart from those of the *Pomaceæ*, and it can also give rise to apple canker. Canker is produced on *Fagus* by both species of the fungus. Finally, it may be noted that *N. coccinea* appears on the smooth, dead bark of a number of trees without causing any canker formation.

Some investigators hold to the old view that the cankers of both pomaceous and non-pomaceous trees are due to the same fungus—*Nectria ditissima* Tul. (Voges, 1913). With regard to the adaptation of the forms of fungi to their environment and the possible modification thereby of their power of attack, it is not difficult to imagine that under special conditions the biological characters of the forms may vary according to the habitat and in such a manner that they can reciprocally deputize one for the other.

Nectria Cucurbitula (Tode) Fr. only attacks conifers, preferably spruce, more rarely fir and pine. The fungus does not produce a real canker—at most, only a one-sided thickening. In consequence of the destruction of the cambium the head of the tree frequently dies back or the tree itself may be killed. Infection of the tree takes place in wet weather, either in autumn or early spring, and follows upon injuries to the bark by hail or frost. On young trees the fungus is produced in rings on the lower parts of the stem. On older trees the white cushions of conidia first appear and later the perithecia arise in these stromata, but there is no formation of cracks or wounds. The bark only dries up and dies in the following summer. Sometimes the affected cambium again becomes active, in which case the dead tissue is cut off by a layer of cork and the spread of the fungus is in this way stopped.

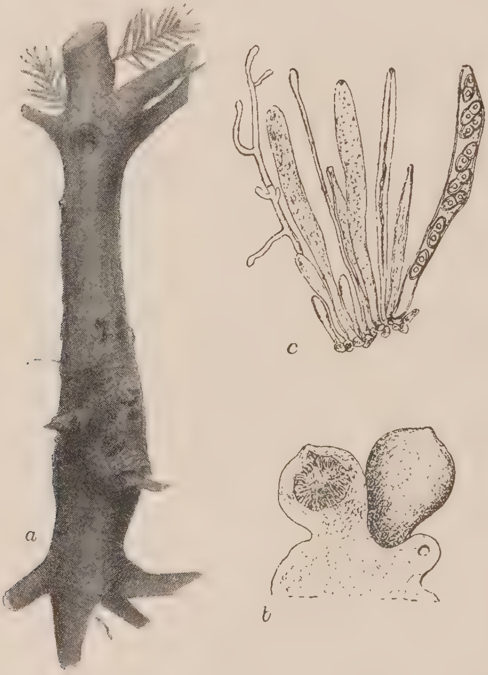

Fig. 256.—*Nectria Cucurbitula.*

a, the lower part of eight-year-old spruce bearing sporophores; *b*, perithecia; *c*, asci and paraphyses. (*a*, after E. Rostrup; *b* and *c*, after E. Prillieux.)

When the outbreak of disease is severe there is no alternative but to fell the tree or cut off the infected branches and burn them.

Nectria cinnabarina (Tode) Fr. appears on numerous deciduous trees and shrubs, preferably those in avenues and parks, more rarely on those growing in woods. The trees most commonly affected are species of the genera *Acer*, *Æsculus*, *Carpinus*, *Fagus*, *Morus*, *Robinia*, *Ribes*, *Tilia*, etc. In Denmark the fungus has been found on fifty different species of trees (Rostrup, 1902).

It was long considered that the fungus only lived and grew upon the dead tissues of the host plant. Later investigations have, however, shown conclusively that the mycelium of the fungus attacks the living cells and causes the death of the tissues and organs that are affected, in spite of the fact that the conidial pustules and the perithecial stromata are only found on the dead bark. During the colder periods of the year the stromata, or conidial pustules (*Tubercularia vulgaris* Fr.), break through the dead portions of the bark; they are about the size of a pin's head, of a flesh or vermilion colour, becoming paler later, and they bear large numbers of conidia which are unicellular, ellipsoidal, and colourless. In the spring, very small, globose, orange-red, darker with age, smooth perithecia develop. At times the development of the perithecia is so considerable that they entirely cover the stroma. The asci contain eight spores, two-celled, which are ripe in late autumn and germinate at any time of the year when the weather is damp. As the conidia, as well as the ascospores, are embedded in a mass of mucilage when they make their appearance in the open, they are not suitable for dissemination by wind, and it is therefore thought that the disease is spread through the agency of insects (Behrens, 1895).

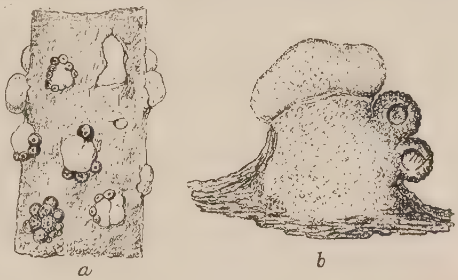

FIG. 257.—*Nectria cinnabarina*.

a, portion of stem with conidial pustules (*Tubercularia*) and perithecia (*Nectria*); *b*, longitudinal section of a group of perithecia. (After E. Prillieux.)

In certain circumstances—probably as a result of large supplies of food or the continued moist state of the stroma—large, usually six-celled, sickle-shaped, so-called macroconidia of the *Fusarium* type (*Fusisporium*) are produced from the stroma. These macroconidia are formed before the individual conidia arise, and they can pass into a resting condition.

The infection of the tree takes place either on the branch, stem, or root, but only when the woody tissue is exposed owing to a wound or injury which may have been caused by the breaking of a branch, by hail, or when the ground was being dug. It is only at such injured places that the necessary amount of mycelium is able to develop. The fungus creeps along the surface of the wood of the wound until it reaches the opening of an injured vessel or a wood cell. Into this opening the fungus enters, develops further, and produces mycelium in the wood. If the mycelium reaches a cell containing starch granules, the cell-contents and starch are broken up and consumed.

This species of *Nectria* does not generally cause the formation of cankers on the stems or branches that are attacked, as the mycelium develops so rapidly that there is not time for layers of callus to be formed at the wounds. In a few rare instances (as in Hanover, 1889), once on *Morus alba* and once on *Broussonetia papyrifera*, the disease has been indicated by depressions in the bark or by canker-like, raised patches.

It is only possible to save a tree attacked by this fungus if the disease is limited to the surface of the branch and the branch is removed close up to the stem. Should, however, the main stem be attacked the tree is doomed and should be cut down so as not to be a source of infection to other trees (Brick, 1892; Wehmer, 1894, 1895; Behrens, 1895; Rostrup, 1902; Beck, 1903).

Polystigma rubrum (Pers.) DC. produces large, round, hyaline, thick patches of a reddish-yellow or bright red colour and with sharply-defined boundaries on the leaves of *Prunus domestica*, *P. insititia*, and *P. spinosa*. On the under surface of the patches are found small, black dots which are the mouths of pycnidia embedded in the leaf tissue. In the course of the winter, perithecia develop in the patches on the fallen, partly-decayed leaves, and from these perithecia, which ripen in April and May, ascospores are ejected and infect the young leaves on the trees. As a preventive measure it is recommended to rake up the fallen leaves in autumn and burn them. The ground below the trees should also be dug over in spring before the new leaves appear (Frank, 1883, 1896). **P. ochraceum** Wahl. produces large, orange-yellow patches on the leaves of *Prunus Padus*.

CHAPTER XIII

DOTHIDEALES

The perithecia are embedded in a stroma, without well-defined walls.

Phyllachora graminis (Pers.) Fuck. produces elongated, black, slightly hyaline patches on the leaves of *Dactylis glomerata*, *Agrostis stolonifera*, and other grasses. The patches appear finally on both sides of the leaf. The leaves turn yellow and wither prematurely.

Plowrightia Trifolii Kil. attacks the leaves of several varieties of clover, particularly *Trifolium incarnatum* and *T. repens*, in fields where the crop is growing luxuriantly. The disease is characterized by small, crust-like spots, black on the under surface and brown on the upper, which bear pear-shaped, two-celled, brown conidia in chains. By means of the conidia the fungus spreads during the growing period, sometimes, in the case of crimson clover, to such a degree that whole fields are destroyed. The production of conidia continues until winter, and from this time onwards the fungus enters upon a new stage. In the tissues of the dead leaves, most of which have fallen by this time, sunken pycnidia arise which develop pycnospores whose function is to spread the disease during the winter. The stage of the fungus upon the living clover plant during the growing period was formerly named *Polythrincium Trifolii* Kunze.

To ensure the reappearance of the disease on the clover plant in the next year, perithecia are produced in the late autumn on leaves that are still green. These perithecia are often found together with pycnidia on the same leaf, and like them are embedded in the leaf tissue ; the perithecia, however, only ripen after over-wintering under a protective layer of soil. The perithecia contain 2–4 asci and each ascus 8 spores which are two-celled. It is from these ascospores, which germinate in the spring, that the disease passes to the new clover crop (Killian, 1923).

Plowrightia morbosa (Schw.) Sacc. gives rise to a disease known in America as Black Knot. The fungus appears on a number of species of *Prunus*, such as *P. angustifolia*, *P. americana*, *P. serotina*, *P. pennsylvanica*, *P. virginiana*, *P. avium*, and *P. Cerasus*, often very destructively. The swellings caused by the fungus are closely grouped together, black in colour, about 1 cm. high, and with a broken, uneven surface. Starting from these knots the mycelium grows into the cambium tissue of the branch. New knots continue to be formed which lead to the death of the branch and finally of the whole stem. The fructifications of the fungus develop on these knots, beginning in May with the conidial stage with conidiophores and pycnidia, later perithecia with asci and narrow paraphyses. The ascospores are hyaline, two-celled, the lower cell very small. To control the disease it is recommended to remove any parts of the tree that are badly affected and burn them, sometimes the whole tree may have to be cut down, in less severe cases the knots should be cut out.

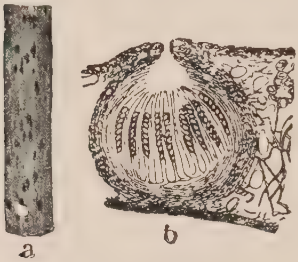

Fig. 258.—*Phyllachora graminis.*

a, piece of leaf of *Triticum caninum* with patches of fungus; *b*, sunken perithecium in section with numerous asci. (*a*, Orig.; *b*, after A. B. Frank.)

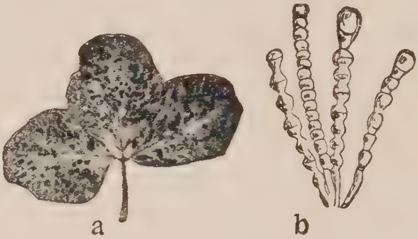

Fig. 259.—*Plowrightia Trifolii.*

a, leaf with spots of fungus; *b*, formation of conidia. (*a*, after E. M. Freeman; *b*, after E. Rostrup.)

Diachora Onobrychidis (DC.) J. Müll. produces black, crust-like patches on the leaves of *Onobrychis sativa* and *Lathyrus tuberosus;* both pycnidia and true perithecia are developed on these patches.

CHAPTER XIV

SPHÆRIALES

PERITHECIA with well-defined walls, with or without stroma.

Blight or Sooty Mould of Cereals

Mycosphærella Tulasnei Jancz.

Cladosporium herbarum Link.

It is not infrequently found about harvest-time in wet years that small, black dots, arranged in rows, appear on the yellowing leaves, stems, glumes, and even on the grain, of rye and wheat, sometimes also on other cereals. Each of these dots is seen on examination to consist of a tuft of dark, olive-coloured conidiophores which abstrict simple or branched conidia at their tips. The mycelium from which the tuft arises may be present on the epidermis of the part attacked, or in either the epidermal or subepidermal cells. It consists of closely-compacted, almost black cells which sometimes form a globular, sclerotia-like structure, a sort of rudimentary ascus. In the form just described, the fungus has been known from earliest times under the name of *Cladosporium herbarum*. It was long considered that plants were only attacked by the fungus when for some reason they were weakened and so not capable of resistance. Healthy plants were held not to be susceptible, but later investigations have shown that the fungus must be regarded as a true parasite of cereals which it is able to attack, especially in the case of wheat plants, in all stages of growth. Thus, seedlings may be killed and older plants attacked in the stem, causing them to produce stunted ears or none at all, whilst later attacks at the time of flowering may result in no grain being formed. The final attack, during the ripening period, causes the grains to be small and misshapen.

By special feeding in artificial cultures it has been possible to impart increased vitality to the fungus and to bring it to a further stage of development. The globular sclerotia have in this way been changed into true perithecia with asci; this stage of development is named *Mycosphærella Tulasnei*.

The fungus *Cladosporium herbarum* appears on many other varieties of plants belonging to different families. On the leaves of peas, yellow or brown patches appear, bearing black tufts of hyphæ which abstrict conidia. The plants dry up from below and only flower feebly; on older plants the fungus appears on the pods. In a late, damp autumn the fungus is found living saprophytically on a great many different plants—herbs, shrubs, and trees.

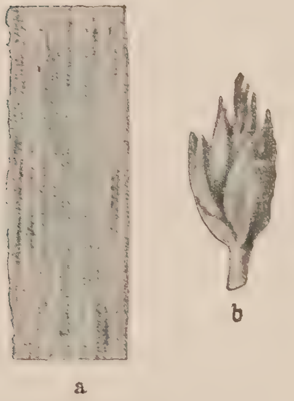

Fig. 260.—Sooty Mould on Wheat (Conidial stage).
a, leaf sheath; and *b*, spikelet with small black dots. (After E. Janczewski.)

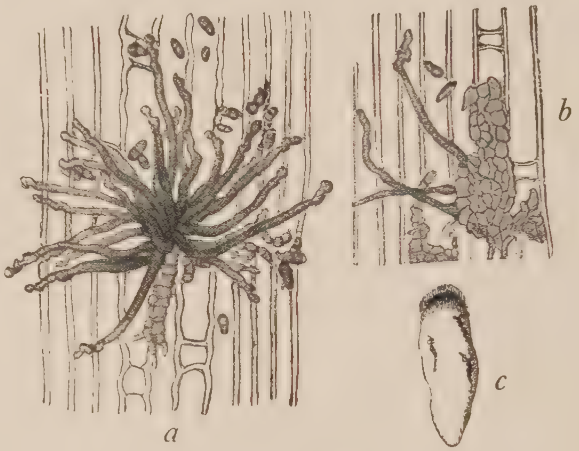

Fig. 261.—Sooty Mould on Rye.
a, leaf sheath with tuft of conidiophores; *b*, leaf sheath with sclerotia-like masses; *c*, diseased grain with sclerotia-like structures on the surface. (*a* and *b*, after E. Janczewski; *c*, Orig.)

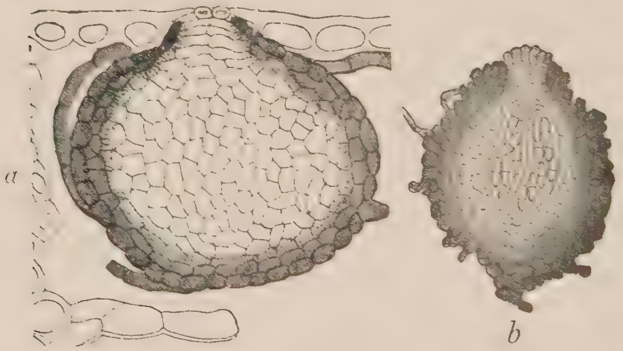

Fig. 262.—Sooty Mould on Rye (*a*) and Wheat (*b*).
Perithecial stage: *a*, sclerotium beginning to form into a perithecium; *b*, perithecium with visible asci. (After E. Janczewski.)

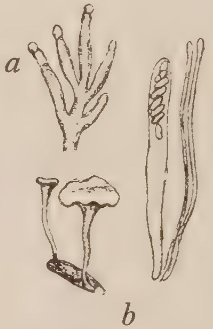

Fig. 263.—*a*, *Endoconidium temulentum*; *b*, *Stromatinia temulenta*. (After E. Prillieux.)

In very wet years a number of other forms of fungi are sometimes found associated with *Cladosporium herbarum* on grains of rye, particularly the conidial stage, *Endoconidium temulentum* Prill. & Del. of the ascomycete fungus *Stromatinia temulenta* Prill. & Del. This fungus gives rise to whitish, cushion-like groups of conidiophores on grains of rye attacked by *Cladosporium herbarum;* the conidiophores produce ovoid conidia at their tips.

It has been noticed in a number of places that rye suffering from this Sooty Mould, or Blight, disease if used for the making of bread, etc., or if fed to animals, has a temporary adverse effect upon health. In the case of human beings the symptoms are headache, lassitude, dizziness, disturbance of the sight, and sometimes a peculiar intoxication similar to that which occurs in poisoning by carbon monoxide. Numerous instances of the serious results which follow upon the use of such diseased rye have been recorded from Sweden (Eriksson, 1883), South Russia (Woronin, 1891), and France (Prillieux, 1897).

The poisonous properties of the infected grain were at first considered to be due to the fungus *Cladosporium herbarum*, but German experiments do not support this view, and it now seems probable that the associated fungus, *Endoconidium temulentum*, is the exciting cause. As an antidote to the poison it has been found that sour milk is valuable, as is also the admixture of oat flour with the rye flour.

Control Measures :

(1) Cereals attacked by blight should be harvested early and the stubble burnt off. (2) Straw badly attacked by the disease should not be used for making manure. (3) Straw and grain should be thoroughly dry before being carried.

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Heart Rot, or Blight, of Beet and Mangel

Mycosphærella tabifica Prill. & Del.

Phoma Betæ (Oud.) Frank.

This disease appears in summer (July-August) in sugar beet and mangel crops, either on single plants or in patches. The youngest leaves in the head of the plant are the first to turn black and die, the disease soon spreads to the next older leaves and from them through the leaf-stalks (which often show light-coloured transverse bands) into the lamina. These leaves also gradually die, and it may happen that towards the end of the summer the plants have lost almost all the original leaves, their place being taken by small rosettes of stunted leaves. The disease spreads at the same time to the beet itself, the outer portions of the largest beets usually being first attacked, and brown, more or less sunken patches produced in the flesh.

Sometimes the disease comes to a standstill, but the beets do not then reach the size and sugar-content of a healthy specimen; generally, however, the infected root decays.

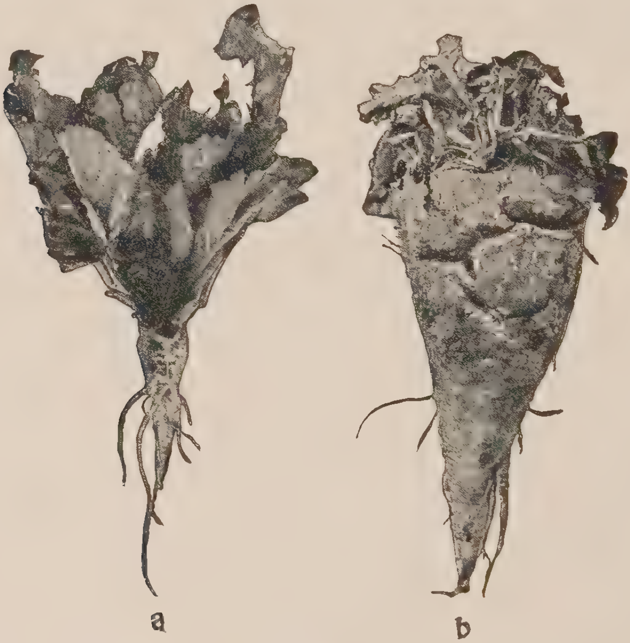

Fig. 264.—Heart Rot of Beet.
a, early stage of disease (in August); *b*, later stage (in October). (Orig.)

The disease attacks mangels as well as sugar beet and varies in severity in different years even in the same locality. It is the general opinion that the disease is favoured by continued dry weather in the summer at the time when leaf growth is at the maximum and the transpiration of water is high in relation to the intake by the roots.

It is a question whether the fungi which have been found in diseased roots are the primary cause, or whether the disease is to be attributed to a weakened condition of the plant induced by intensive cultivation. Numerous investigations of recent date point strongly to some species of fungus being the primary cause of the disease, and in the first place *Phoma Betæ*. This fungus—also named *Phoma sphærosperma* Rostr. and *Phyllosticta tabifica* Prill. & Del.—appears on the infected part of the root in the form of small, dot-like pycnidia within which hyaline, ovoid pycnospores develop. In a moist environment these spores are extruded in long, gelatinous tendrils from the apical opening of the pycnidium. This stage of the fungus is also found on the fructifications of seed-beets even in the ripe seed-capsules, also on roots that are stored in clamps or cellars.

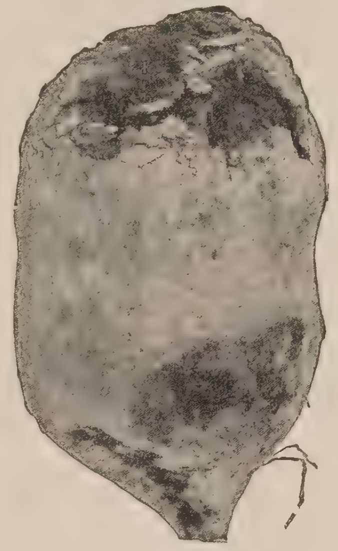

FIG. 265.—Heart Rot on Mangel. (Orig.)

In late autumn, when the conditions are favourable, a further stage of the fungus sometimes appears in fields of beet. This stage (Leaf Spot), which is named *Mycosphærella tabifica* and also *Pleospora putrefaciens* Frank, is in the form of small, black, dot-like perithecia on the older leaves and leaf-stalks. The asci in the perithecia each contain eight multiseptate spores. In wet weather the diseased patches on the leaves quickly decay.

There are other fungi besides *Phoma Betæ* which are concerned in the decay of roots suffering from Heart Rot, the most important being: *Pythium de Baryanum* Hesse, *Aphanomyces lævis* de By., and *Rhizoctonia* sp.

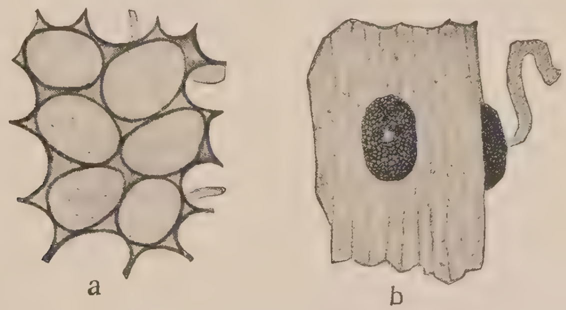

FIG. 266.—*Phoma Betæ*.
a, cell tissue with mycelium; *b*, pycnidia, from one of which a tendril of conidia is being extruded. (After G. Linhart.)

Finally, it has been suggested that the cause of the disease is a slime fungus *Myxomonas Betæ* Brz. which in the beginning lives symbiotically with the plasm of the host cell and later develops as a parasite (Brzezinski, 1906). Such a condition would be similar to the latent stage of development described previously as "Mycoplasm" in the case of certain rust- and downy mildew-fungi. The existence of such a slime fungus has, however, been disputed (Trzebinski, 1907; von Faber, 1908; Peters, 1913). It is also possible that under the name of Heart (or Root) Rot several diseases due to different agents may be included.

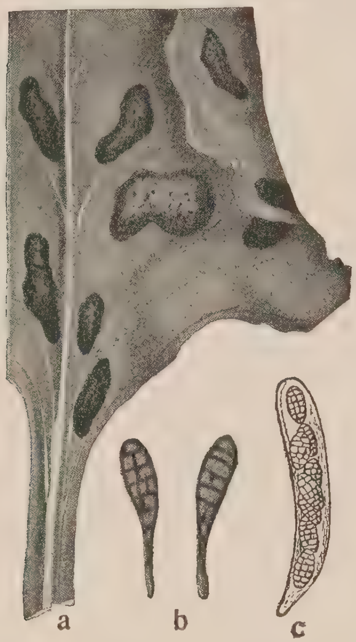

Fig. 267.—Leaf Spot Stage of Heart Rot.

a, leaf with spots; *b*, conidia; *c*, ascus with eight spores. (*a* and *b*, after O. Kirchner and H. Boltshauser; *c*, after A. B. Frank.)

The disease can be traced back to the middle of the eighties of the last century when it caused much damage in Brandenburg and Silesia. In 1892 it was observed and described in France and the following year it appeared in almost all the beet-growing countries of Europe.

Control Measures:

(1) Deep working of the soil should be practised so that drought may be withstood. (2) Manures that induce too rapid growth should be avoided. (3) Sowing should not be too early lest the plants suffer from summer drought during their most critical period. (4) Leaves of diseased beets should not be thrown on the manure heap. (5) Beets should not be grown for at least four years on a field that has produced a diseased crop. (6) Resistant varieties should, as far as possible, be grown.

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Mycosphærella brassicicola (Duby) Lindau produces small, round spots, brownish-grey in the centre with an olive-green margin, on the leaves of the cauliflower. On the outer leaves the spots may be so numerous that they almost entirely cover the surface and cause the leaf to die. On the inside leaves only a few spots appear and they give rise to a yellowing of the leaf. At the edges of the spots very small dots can be seen; these are the openings of the pycnidia which are embedded in the leaf tissue. From the inner walls of these pycnidia are developed conidia which spread the disease. The fungus in this stage of development is named *Phyllosticta Brassicæ* West. Immediately below the epidermis of old, dead leaves perithecia are found; they are at first spherical, closed cell-masses, but they soon break through the epidermis and develop asci. It is in this *Mycosphærella* stage that the fungus hibernates. In Australia and America this fungus appears to be of greater economic importance than in Europe (Osmun and Anderson, 1915; Weimer, 1926).

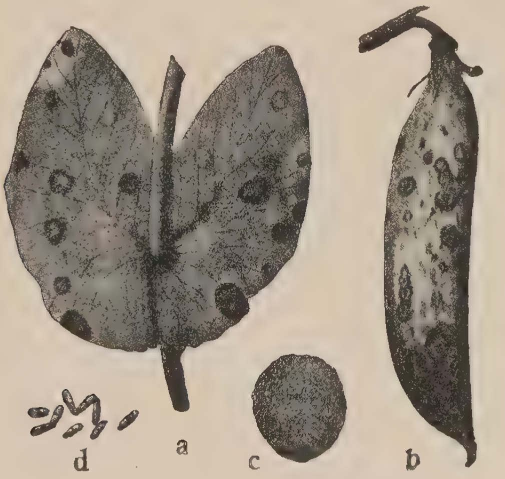

FIG. 268.—Conidial Stage of *Mycosphærella pinodes*. *a*, bracts, and *b*, pod with spots; *c*, pycnidia; *d*, conidia. (After O. Kirchner and H. Boltshauser.)

Mycosphærella pinodes (Ber. & Bl.) Niessl. The conidial stage is named *Ascochyta Pisi* Lib. and produces yellow, brown-edged spots with small, brown to black pycnidia in the middle. These spots appear on the leaves, stalks, and fruits, sometimes also on the seeds of peas, tares, lucerne, and sainfoin. Perithecia arise on the stems and dead fruits. Sometimes different sorts of peas growing near to each other are attacked to a very different extent and transmission of the disease by means of the seed is suspected. The disease is sometimes responsible for a considerable reduction in the crop. Peas harvested from diseased plants should not be used for seed (Stone, 1913 ; Arnaud, 1913 ; Gilchrist, 1926).

Mycosphærella exitialis Mor. and **M. basicola** Frank appear on the leaves and sheaths of wheat, rye, and barley ; they give rise to elongated, pale streaks with small, brown dots. **M. recutita** Fr. produces long, parallel, grey streaks with black dots arranged in rows on the leaves of cocksfoot grass.

Læstadia microspora Awd. has been found on oats in Denmark. The leaf-sheaths, especially the lower ones, are covered with black dots. The plants remain small and the panicles develop imperfectly.

Sphærulina Trifolii Rostr. produces numerous, small, round spots on the upper surface of the leaves of white clover. The spots are light brown in colour, surrounded by a purple margin and covered with small, black dots. This fungus has been observed in Denmark, Germany, and America (Hopkins, 1923).

Pleosphærulina Briosiana Bub. gives rise to small, brownish-red or larger light brown spots on the leaves of lucerne. The larger spots occur mainly on the tips and margins of the leaves and enclose the asci of the fungus. The fungus has been observed in Austria and America (Bubák, 1909 ; Miles, 1925).

Foot Rot or Take-all of Cereals

Leptosphæria herpotrichoides de Not. and *Ophiobolus herpotrichus* Fr.

This is a disease which is found on wheat, rye, and barley, sometimes also on some wild grasses such as *Agropyrum repens* and *Aira cæspitosa.* The causal agents of the disease are the above-mentioned species of fungi ; in Europe it is the *Leptosphæria* fungus that takes the first place. In general, both fungi are regarded as true parasites which are capable of attacking a perfectly healthy plant. Some investigators, however, consider them to be semi-parasites—that is to say, fungi which can only invade plants that are weakened from other causes.

In Europe *Leptosphæria herpotrichoides* appears to play the chief part in producing the disease ; it attacks wheat and rye preferably and causes the stems of the plants to bend and break off about the time of ripening. The fungus attacks the lowest internode of the stem inside the leaf-sheath and causes it to turn brown, whilst upon it are sometimes found a black, superficial layer or scattered groups of hyphæ. The fungus permeates the cell tissues of the stem and prevents the supporting elements from being developed normally. The stem is, in consequence, weakened at the point of attack and is easily broken off by wind or some other external factor. The stems may thus come to lie upon the ground in all directions; those which break off early are without ears, but if ears have formed they are small and the grain shrivelled.

In damp situations there are developed long, multiseptate structures which have been regarded as the conidia of the fungus and compared with *Dictyosporium opacum* Cool. & Harl. The perithecia have a drawn-out beak and are often covered with hairs. The spores are long and pointed with numerous (3–8) septa; they sometimes germinate inside the ascus.

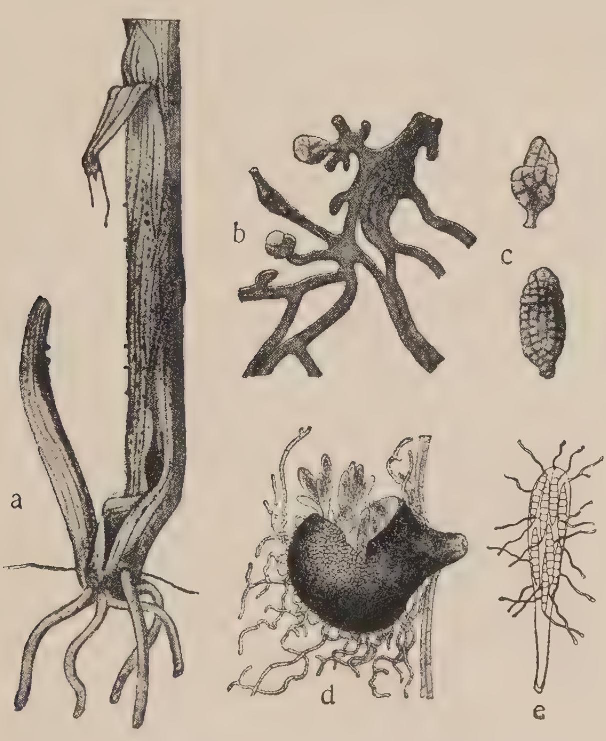

FIG. 269.—*Leptosphæria herpotrichoides.*

a, lower part of wheat plant with perithecia; *b*, mycelium on the surface of the stem; *c*, conidia; *d*, ruptured perithecium; *e*, ascus with germinated spores. (*a* and *d*, after E. Krüger; *b*, *c*, and *e*, after L. Mangin.)

Cold, wet weather in May and June appears to favour the development of the disease. Possibly, too, the weather conditions of the year in which the seed was grown may have an influence, particularly if through drought the crop ripened prematurely. When climatic conditions are unfavourable, the development of the plant is checked and its power of resistance to the fungus is lowered. On this account the disease is most prevalent on cold, stiff soils. A too-luxuriant growth of the wheat crop in a mild winter, as well as late night frosts, must also be considered to further the disease.

The disease has been known in France since the middle of the last century; in England in 1861 on *Aira cæspitosa* and in 1884 on wheat; in Italy in 1875 on *Agropyrum* sp. and in 1886 on wheat. In 1894 the rye-fields in Northern Germany were heavily attacked, in some cases the loss of crop being as much as 75 per cent. In 1903 and 1904 the fungus appeared in most of the other European countries, and it is now regarded as one of the commonest and most dangerous of cereal pests, especially on wheat.

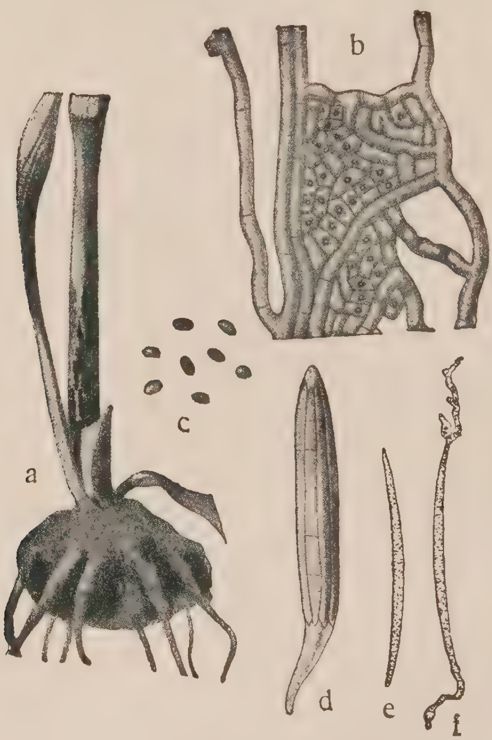

Fig. 270.—*Ophiobolus herpotrichus.*

a, lower part of early ripening wheat plant: black streaks of fungus at base of stem, soil adheres to root; *b*, mycelium; *c*, conidia; *d*, perithecium; *e*, spore; *f*, germinating spore. (*a* and *d*, after A. B. Frank; *b*, *c*, *e*, and *f*, after L. Mangin.)

The different varieties and strains of wheat are affected to a varying degree by the disease. In Sweden it has been found, for example, that the older, native sorts and their descendants are more susceptible than the newer sorts.

Together with the fungus described above there is usually associated another—*Ophiobolus herpotrichus*, sometimes called *O. graminis* and *O. herpotrichoides*—which attacks wheat chiefly, also rye and barley. The fungus forms a black coating on the lowest internode of the stem, inside the leaf-sheath, and often causes the whole of the lower part of the plant to become black. The roots are also attacked and are turned black for the greater part of their length. When affected plants are drawn from the ground the soil generally adheres to the roots. Some of the plants remain short and have poorly developed ears; others grow to a greater length but soon wilt, the plants becoming " sooty " even up to the ears. The grains remain small and shrivelled.

A black powder, in which very small, unicellular, ovoid spores are said to have been distinguished, is sometimes found on the diseased parts of the stem and has been regarded as a conidial stage (a *Coniosporium* form) of the fungus, but whether this view is correct has not been decided.

The perithecia, which appear later in the autumn, are globose with a curved neck and are sunk in the tissues; they are smooth in the case of *O. graminis* and hairy in the case of *O. herpotrichoides*. The spores are eight in number, thread-like, colourless, those of *O. herpotrichoides* being narrower and longer than those of *O. graminis;* inside the spores are rows of small drops and several septa.

This disease occurs, as a rule, only in patches in the cornfield, and in this way differs from the attack of *Leptosphæria herpotrichoides* which appears uniformly over the whole field. In Sweden, *O. graminis* has been very destructive to certain varieties of wheat, whilst in Italy, France, and Germany, *O. herpotrichoides* is common. In Italy, a similar disease has been observed on couch grass (*Agropyrum repens*) and in Australia on brome grass (*Bromus sterilis*).

In America, Foot Rot or Take-all has been known since the beginning of the present century. The causal fungus is there stated to be *Ophiobolus cariceti* (B. & Br.) Sacc. which is considered to act alone. In 1921, in New York State, the disease was found on 78 out of 224 wheat-fields and the damage in one case amounted to 20 per cent. The disease appears to be spread mainly by infected soil or by pieces of diseased straw containing perithecia being mixed with healthy soil. The fungus is favoured by an alkaline reaction of the soil, whilst an acid reaction checks it. No difference in susceptibility of individual varieties of wheat was observed in the case of cultivated sorts. By artificial inoculation the disease has been transmitted in the greenhouse to one or more species of the genera *Agropyrum*, *Bromus*, *Elymus*, *Festuca*, *Hystrix*, *Lolium*, *Phalaris*, etc.

In Australia, Foot Rot has been known since 1852, and since the beginning of this century the causal fungus has been said to be *Ophiobolus graminis* Sacc.

Control Measures :

(1) Early and deep ploughing of the stubble. (2) Harrowing the autumn-sown cornfields in spring. (3) After harrowing, an additional dressing of nitrate of soda. (4) The frequent growing of the same variety of cereal in a field should be avoided. (5) The use of basic slag as a manure is recommended. (6) Excessive use of nitrogenous manures should be avoided.

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Dilophia graminis (Fuck.) Sacc. This fungus attacks a large number of species of grasses. It possesses conidia which are either free or sunk in a globose receptacle, and perithecia which contain asci and ascospores. In the free conidial stage, long, reddish-brown spots, surrounded by a clear margin, appear on the leaves. The middle of the spot is chalky-white and bears spindle-shaped, colourless conidia which have three septa and are usually furnished with a few delicate threads at each end. This conidial form, which is also named *Mastigosporium album* Riess, is found on *Phleum pratense*, *Dactylis glomerata*, *Alopecurus pratensis*, etc. The form in which the conidia are enclosed in pycnidia is known also as *Dilophospora graminis* Desm. and gives rise to greyish-black crusts, white on the inside. The conidia are long with a tuft of fine hairs at each end. This form brings about the distortion of the leaves of *Holcus lanatus*, *Alopecurus pratensis*, and other grasses.

In England, France, and Switzerland this fungus has been very injurious to winter corn, causing the ears to become thin, black, and misshapen with only a few grains.

Gibellina cerealis Pass. This fungus attacks wheat only. In spring, plants which have reached a height of 10–12 inches are observed to turn yellow and begin to wilt. On the haulms and leaves, more frequently on the leaf-sheaths, greyish-brown circular spots appear which at first have a well-defined margin but later elongate and coalesce. The spots are covered with a thick felt formed of much-branched, thick, hyaline hyphæ which abstrict ovoid conidia arranged in chains. The leaves, especially the lower ones, become dry and in-rolled ; the ears either do not form or, if formed, remain quite empty. Black dots, arranged in rows or groups, appear in the grey felt, chiefly on the leaf-sheaths. These dots are the mouths of the flask-shaped perithecia which are embedded

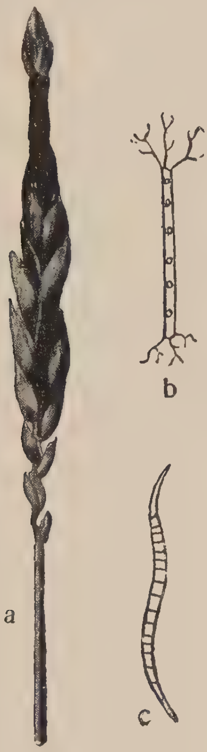

Fig. 271.—*Dilophia graminis*.

a, deformed ear of wheat ; *b*, conidium ; *c*, ascospore. (*a*, after O. Kirchner and H. Boltshauser ; *b* and *c*, after E. Rostrup.)

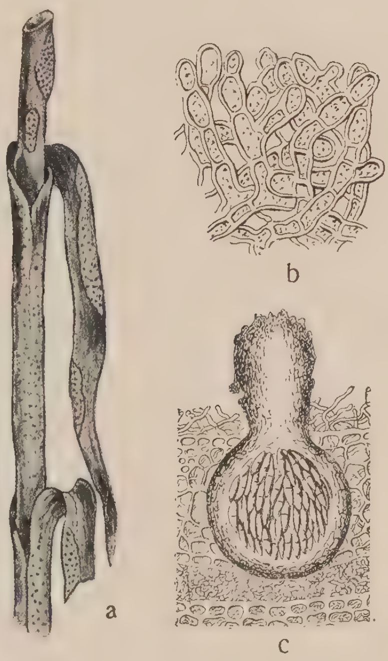

Fig. 272.—*Gibellina cerealis* on Wheat.

a, stem with spots of felt of fungus ; *b*, surface mycelium which produces conidia ; *c*, sunken perithecium. (*a*, after G. Massee ; *b* and *c*, after F. Cavara.)

in the leaf tissue; the asci in the perithecia contain each eight spores arranged in two rows. The wall of the ascus is thin and soon dissolves, so that the spores lie free in the perithecium. The spores appear to over-winter in the soil and infect the young plants in the following year.

This fungus was first observed in wheat-fields near to Parma (Northern Italy) in 1883 and described in 1886; in 1891 it was again found (near Florence) and later in Hungary and France. The disease does not seem to appear each year (Cavara, 1893).

As control measures it is recommended to collect and burn all infected haulms early in summer and not to grow wheat in the following year on a field that has produced a diseased crop.

Leaf Stripe of Barley

Pleospora trichostoma f. sp. *Hordei* Died.

Helminthosporium gramineum Rab.

The disease appears on barley plants early in summer—the end of May or the beginning of June—in the form of one or more long, narrow stripes on the leaf-blade. At first the stripes are pale yellow, afterwards black with a narrow yellow border. The number of stripes gradually increases until the leaf may be almost covered; it then begins to split—especially at the tips—into long threads. The plant ceases to grow, ears are not formed or only a single one which is empty. Diseased plants occur singly, not in large patches nor in the whole field.

A black, dusty powder soon appears on the stripes and finally covers the whole plant. The powder consists of short hyphæ which, singly or in groups, push through the stomata of the leaf and produce conidia. The conidia are cylindrical with several—usually 4–5—septa, at first yellow, later olive-coloured or brown; they adhere very loosely and are spread by the wind. When conidia are carried from the leaf to the open flower of barley and there germinate, for example, on the moist stigma, they produce a mycelium which spreads through the parenchymatous tissue of the seed coat and changes there into a resting mycelium, without affecting to a noticeable degree the ripening of the grain. When seed barley is sown in spring the resting mycelium germinates at once, if moisture is available, and penetrates into the young seedling as it grows. In this way the disease is carried on from year to year by means of the seed. The conidia seem to play practically no part in the spread of the disease from leaf to leaf in summer and from plant to plant.

The fungus just described is usually named *Helminthosporium* *gramineum*, and it is followed by another form—*Pleospora trichostoma*—which lives saprophytically on the dead haulms and occurs on stubbles where diseased plants have grown. It appears as small, semi-globular sclerotia, black on the outside and furnished with stiff, dark bristles that have a violet sheen. The sclerotia remain unchanged over the winter, but in spring they either give rise to perithecia, in which case the 4–5 outer layers of pseudoparenchyma serve as a wall and the rest is used for the development of the ascus, or, when in a moist atmosphere, conidia are formed on the surface of the perithecium. Neither the ascospores nor the conidia can infect a fully-developed plant, and consequently stripe disease does not spread in barley-fields during the summer. Artificial inoculation experiments, made indoors by placing small pieces of barley leaf carrying stripe disease upon young barley plants, led to infection as soon as the first leaves were a few inches above ground.

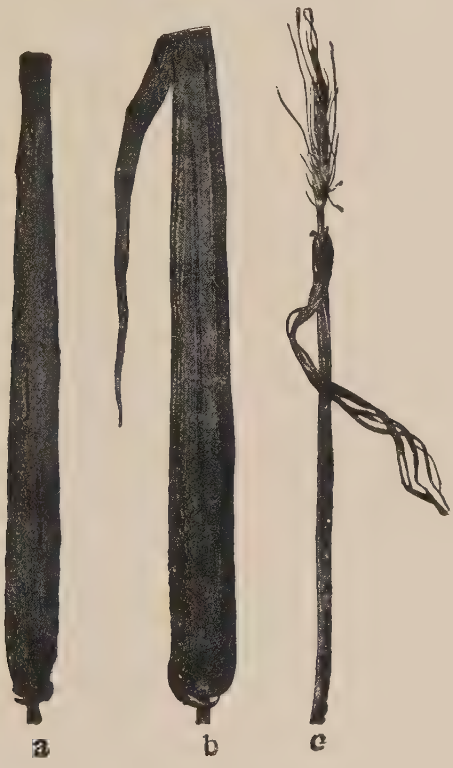

Fig. 273.—Leaf Stripe of Barley.

a, young and *b*, old stage of disease on leaves ; *c*, tip of diseased haulm. (*a* and *b*, Orig. ; *c*, after F. Kölpin-Ravn.)

The fungus **Pyrenophora Tritici-repentis** (Died.) Dresh. on *Agropyrum repens* is closely related to the one just described.

Stripe disease of barley occurs throughout Mid-Europe and also in the Scandinavian countries and North America. In some years in Denmark, 20–40 per cent. of the crop has been destroyed.

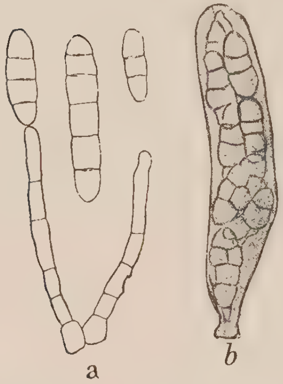

FIG. 274.—*Helminthosporium gramineum.*

a, mycelium with conidia ; *b*, ascus with eight spores. (*a*, Orig. ; *b*, after E. Noack.)

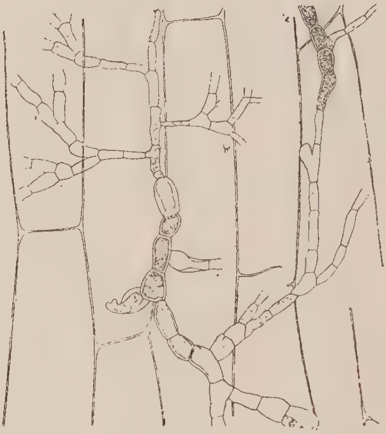

FIG. 275.—*Helminthosporium gramineum.* Germinating resting mycelium on the epidermis of the fruit wall. (After E. Vogt.)

Control Measures :

(1) Seed should be obtained from a crop which showed no disease in the previous year. (2) If such seed is not obtainable, then that which is to be used should be disinfected with an appropriate fungicide. (3) Seed should not be sown too early nor in cold weather.

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Net Blotch of Barley

Pyrenophora teres (Died.) Drechs.

Helminthosporium teres Sacc.

This disease differs from Stripe Disease in having short, separated patches in place of stripes on the leaf-blade and by the absence of splitting; nor does the lower part of the leaf lose its elasticity, but retains its normal shape and position. The ear also develops more or less normally, as do also the grains, except for a bluish-brown colour at the tips.

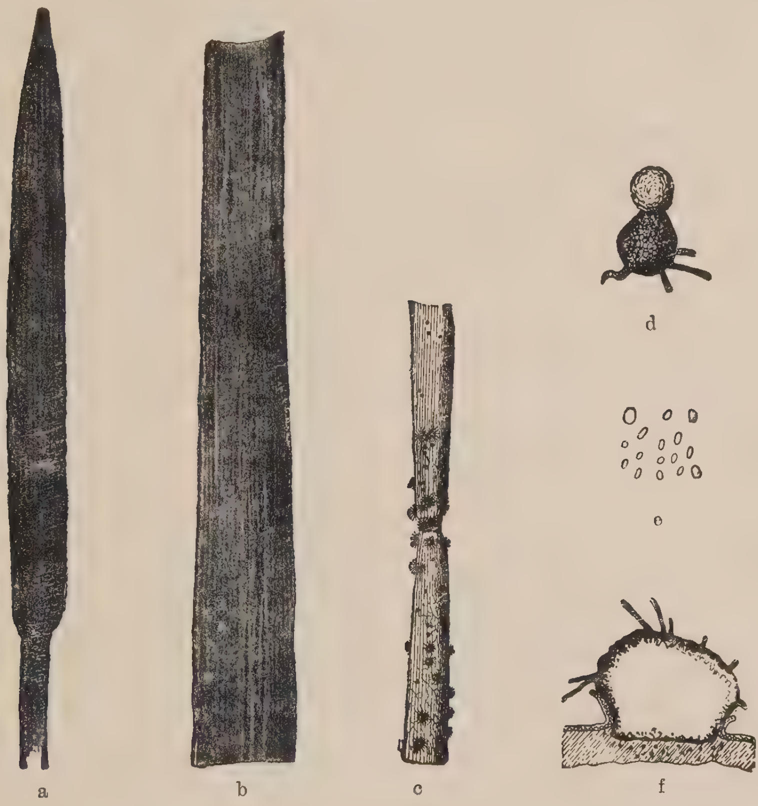

Fig. 276.—Net Blotch of Barley.

a, primary stage on young leaf; *b*, secondary stage on older leaf; *c*, haulm with pycnidia (black dots) and sclerotia, larger structures with bristles; *d*, pycnidium; *e*, conidia; *f*, sclerotium in section. (After F. Kölpin-Ravn.)

The outbreak of the fungus is in large areas or over the whole field, and when weather conditions are favourable the disease may become epidemic at any period of growth of the crop; damp weather favours its development.

Two stages of the disease are distinguished—the primary one on the young leaves, due to infected seed, and the secondary one on the older leaves which are probably infected by the diseased leaves of the primary attack. By means of conidia, the disease spreads during the growing period from leaf to leaf. The spots on the leaves are covered with a greyish-black powder composed of hyphæ and conidia.

The fungus just described is named *Helminthosporium teres* and this form is followed by another which lives saprophytically on pieces of dead straw. This second form gives rise to very small, wart-like pycnidia, which produce small, spherical conidia, also to larger sclerotia covered with bristles. The sclerotia hibernate and continue their development in the following spring.

The disease was first observed in 1881 in Italy, about ten years later in Germany, and a further ten years later in Denmark. It has been found in twenty-one of the American states, in districts where barley is an important crop, and the losses which it causes are serious.

The primary outbreak can be prevented by dressing the seed with a fungicide, but such treatment appears to have no effect upon the secondary outbreak of disease.

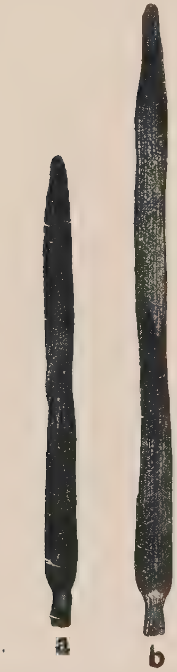

Fig. 277.—*Helminthosporium Avenæ*. (After F. Kölpin-Ravn.)

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Closely related to the above are: (1) **Helminthosporium Bromi** Died. (*Pyrenophora Bromi* Died.) on *Bromus asper* and *B. inermis* in Germany and America. (2) **H. Avenæ** Eid. (*Pleospora Avenæ* Died.) on oats in Germany, Holland, Italy, etc. (3) **H. sativum** Pam., King & Bak. on barley, wheat, rye, and numerous other cereals in almost all European countries, but especially in America where it is very destructive (Christensen, 1922; Stevens, 1922; Dosdall, 1923; Drechsler, 1923).

In addition, eighteen other *Helminthosporium* fungi have been distinguished living on various grasses, and they are regarded and described as separate species (Drechsler, 1923).

Leaf Spot of Cereals

Leptosphæria Tritici (Gar.) Pass.

This fungus attacks all species of cereals. It was first observed in Italy on wheat, afterwards in Germany on wheat, barley, and oats, in Denmark on barley and rye, and in Sweden on barley. Up to the present it is not known whether the same form of fungus attacks all cereals or whether there are specialised forms. To the naked eye, the disease appears as small, black dots on the leaves, the dots being distinctly visible if the transparent leaf-sheath is held against the light. The dot-like structures are spherical perithecia which contain a number of asci, each ascus with eight four-celled spores. In addition to the black dots there are also present on the leaves and leaf-sheaths about an equal number of clear, brown dots which contain numerous rod-

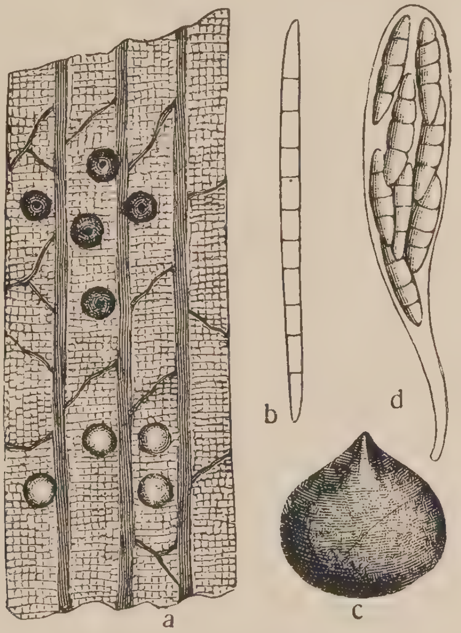

Fig. 278.—*Leptosphæria Tritici.*
a, leaf sheath with light-coloured pycnidia and dark perithecia; *b*, conidium; *c*, perithecium; *d*, ascus with eight spores. (After E. Rostrup.)

shaped, segmented conidia; it is not known whether these conidia are an earlier stage of development of the fungus.

In severe outbreaks of the disease the crop may be considerably reduced, as the ears ripen prematurely and the grains remain small and light. Wet weather in conjunction with a high temperature and little wind favours the spread of the disease.

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Leptosphæria exitialis (Mor.) Web. appears on a number of different varieties of wheat, all parts of the plant being attacked. The fungus can also infect rye and *Poa pratensis* but not other species of cereals and grasses. The conidial stage—formerly known under the name of *Septoria nodorum* Berk. (1875) and *Phoma Hennebergii* Kühn (1877)—appears on the glumes of wheat as large, irregular, greyish-brown or chocolate-brown spots. On these spots, small, dot-like pycnidia are present which contain a large number of septate conidia. The perithecial generation has been found on wheat in July as subepidermal, almost globular, depressions which contain asci with eight spores. When the attack is severe the plants are stunted, the haulm becomes soft, and the ears develop to only half the normal size. The disease is known in many European countries —England, Germany, Italy, and Sweden—and in all wheat-growing districts of America, especially in the southern and eastern central states (Kühn, 1877; Weber, 1922).

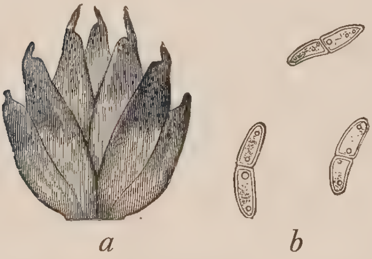

Fig. 279.—*Septoria nodorum* on Ears of Wheat.

a, spikelet with spots on the glumes; *b*, conidia. (Orig.)

Leptosphœria circinans (Fuck.) Sacc.

Rhizoctonia Medicaginis DC.

This disease makes its appearance on lucerne in June or July, circular patches of plants becoming yellow and wilted. The leaves and stems of the plants bear no indication of disease, but if a plant be removed from the soil it is seen that the root is more or less surrounded by a violet-red felt of hyphæ. The part of the mycelium which lies within the tissues of the root obtains the food required by the fungus, whilst the part on the surface of the root serves for its propagation. In the surface mycelium numerous round, brownish-black bodies are found, and these are similar to the pycnidia or perithecia of other fungi. The bodies, however, consist only of interwoven hyphæ and are to be regarded as sclerotia. They are of two kinds, the first being small and black with a brown outer layer and an inner colourless one, the second being larger, brown in colour, and with a brown outer layer and a white inner layer.

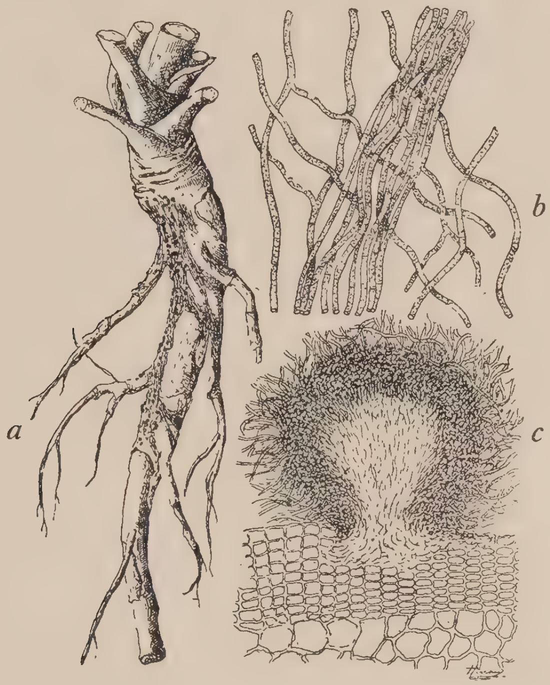

Fig. 280.—*Rhizoctonia Medicaginis.*

a, root of lucerne covered with *Rhizoctonia* felt in which sclerotia are embedded; *b*, mycelium; *c*, sclerotium. (After T. Ferraris.)

In this stage of development the fungus is named *Rhizoctonia Medicaginis* DC. (*Rh. violacea* Tul.).

In addition to the stage just described there is another in which true perithecia are found. Small, black, wart-like growths, grouped together and resembling in shape a sugar-loaf or pear, are formed on the surface of the upper parts of the root and particularly in places where the coating of mycelium is thin. The perithecia, which burst at the sides, contain numerous eight-spored asci; the spores are arranged either in one row or irregularly. Each spore consists of four cells, the two middle ones being large, thick-walled, and of a yellowish-brown colour, the end cells smaller and pale yellow. After the ascospores have over-wintered in the open they begin to germinate in April.

This disease of lucerne was first described under the name "Luzerne couronnée" in France in 1815. In 1861 it was reported in Germany where a number of serious outbreaks have occurred in various parts of the country, *e.g.* in 1893–94 and again in 1900–1910. In Austria and Italy the disease has been known since 1910 and in Sweden since 1911. When the outbreaks of disease are severe, large fields of lucerne may be completely destroyed in 2–3 years.

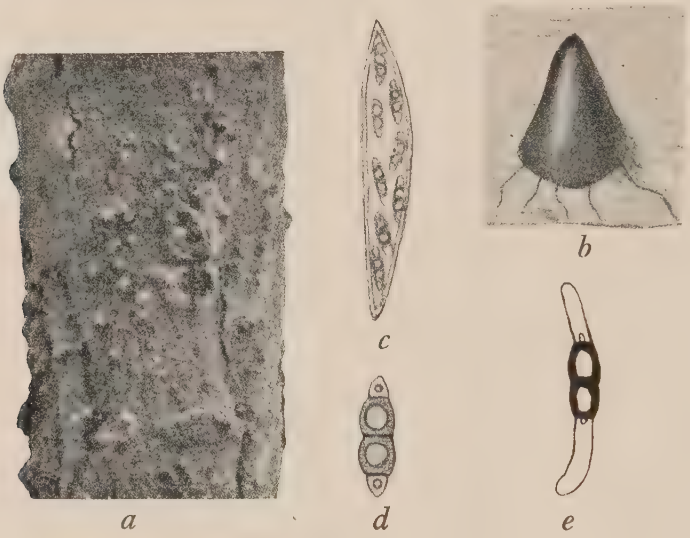

FIG. 281.—*Leptosphæria circinans.*

a, portion of upper part of root of lucerne with coating of mycelium and groups of perithecia ; *b*, perithecium ; *c*, ascus ; *d*, ascospore ; *e*, germinated ascospore. (Orig.)

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Closely related to the above disease, if not identical with it, is the **Root Felt Disease of Clover,** *Rhizoctonia violacea* DC., forma *Trifolii.* This disease agrees in all essential details with that of lucerne, save that up to the present no perithecial stage of the fungus has been observed. In addition to this difference in development there is also the irregularity with which the two forms appear together in different places. In view of these facts the identity of the two forms consequently remains an unsolved problem. It was found, for instance, in Germany in 1893 that the fungus was present on clover in 6 places and on lucerne in 27 places; in 1894 on clover in 8 places and on lucerne in 76; in 1903–04 on clover in 1 place only each year and on lucerne in numerous places. This difference in the behaviour of the fungus on clover and on lucerne is very striking, as it is known that during the two last decades of the previous century *Rhizoctonia violacea* appeared so generally on *Trifolium pratense* in Denmark (and to some extent also on *T. hybridum* and *T. repens*) that most of the clover fields were found, on examination, to be considerably damaged.

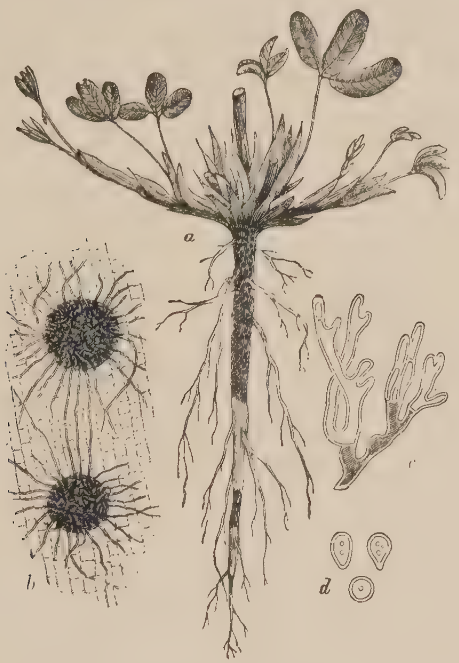

Fig. 282.—Root Felt Disease of Clover.

a, diseased clover plant; *b*, mycelium with pycnidia-like balls of mycelial threads; *c*, portion of mycelium; *d*, conidia. (After E. Rostrup.)

In individual cases a similar disease has also been observed on *Medicago lupulina*, *Ornithopus sativus*, *Vicia Faba*, as well as on lupines.

Root Canker of Carrot

Leptosphæria Rostrupii Lind.

Phoma Rostrupii Sacc.

This disease appears in its conidial stage—*Phoma Rostrupii*—towards the end of summer on the uppermost part of the root, round the base of the leaves, and also on that part of the carrot which is just below the ground. Sunken, grey patches occur in which are small wart-like pycnidia containing a large number of ellipsoid conidia held together in a gelatinous mass. In wet weather, the ripe conidia are extruded from the opening of the pycnidium in the form of long, flesh-coloured tendrils.

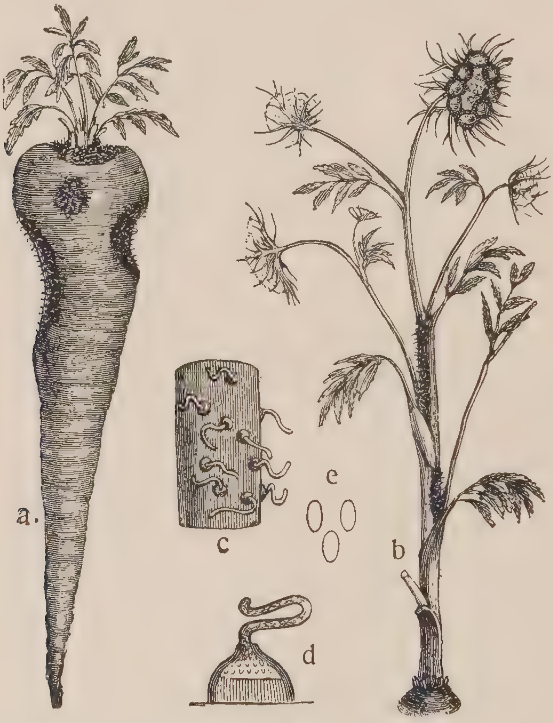

FIG. 283.—Root Canker of Carrot.

a, Carrot with sunken patches on which pycnidia are visible; *b*, flowering plant with similar patches; *c*, portion of stem with conidial tendrils; *d*, pycnidium in section; *e*, conidia. (After E. Rostrup.)

When infected carrots are stored for the winter the disease spreads to healthy roots.

The production of seed from carrots is seriously interfered with by this disease, for if an infected root be planted the fungus spread upwards and gives rise to brown streaks, covered with pycnidia, on the stem. These pycnidia are similar to those which were present on the root of the carrot in the previous year. When the plant is about to flower the carrot may be already rotten, which causes the stem to become flaccid and the plant dies without producing seed.

Diseased carrot stems that have over-wintered out of doors bear hundreds of dot-like perithecia in spring. These perithecia are formed on the patches on the stem, and as they contain asci the fungus has been referred to the genus *Leptosphæria*. The spores, when freed from the ascus, are the cause of fresh outbreaks of disease.

This disease has been known for a long time in Denmark and it has there led to much loss. Some sorts of carrots are attacked to a greater degree than others, the most serious outbreaks generally occurring on light, sandy soils containing peat.

Control Measures :

(1) All diseased roots, even those that are only slightly affected, should be destroyed when the crop is harvested. (2) Stored carrots should be examined from time to time and the diseased ones removed. (3) Only absolutely sound roots should be used for the growing of seed. (4) Varieties of carrots that experience has proved to be resistant to the disease should be selected. (5) Seed should come from a healthy crop. (6) The soil in which carrots are grown should be well prepared and contain adequate food-material for the crop. (7) A field on which a diseased crop has grown should not be used again for carrots for several years.

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Leptosphæria Napi (Fuck.) Sacc.

Sporidesmium exitiosum Kühn.

This fungus attacks a number of plants of the cabbage family such as rape, turnip, kohl-rabi, cabbage, cauliflower, etc. It appears preferably on the stems, flower stalks, and seed pods, sometimes also on the leaves. The fungus forms oblong or roundish, black to brown spots on which hyphæ grow, each hypha producing a conidium. The conidia are elongated, brown, with numerous septa ; the point is drawn out to a long beak. In this stage the fungus is named *Sporidesmium exitiosum*.

Perithecia, which are regarded as a later stage of the fungus, appear on the dead plants, the perithecia are globose with a short, drawn-out mouth. The asci contain long, yellow spores with numerous septa.

The disease does most damage to the seed pods, which become bent, and as they open prematurely the seed does not ripen completely. As protection against the disease it is best to cut rape early, before it is ripe, and then leave it to ripen off.

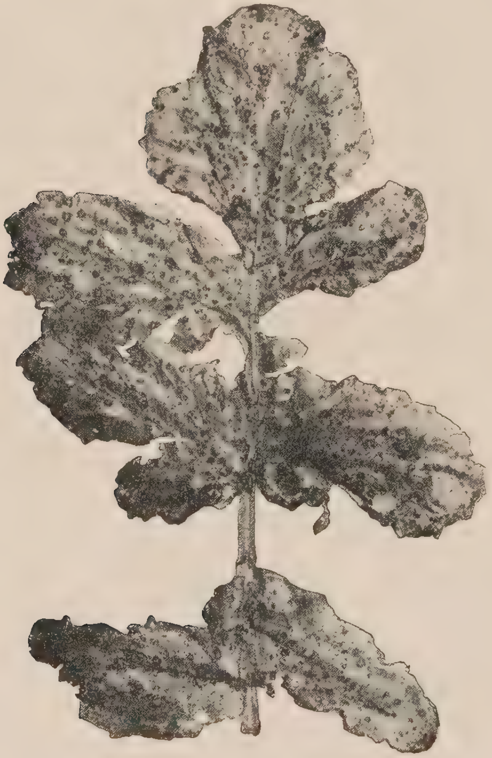

Fig. 284.—*Leptosphæria Napi* on Leaf of White Turnip. (Orig.)

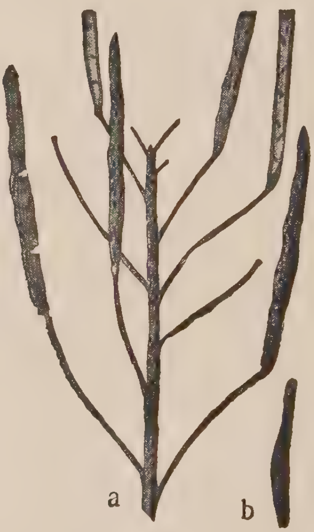

Fig. 285.—*Leptosphæria Napi* on Fructification of Rape.

a, fructification with black fungus spots ; *b*, conidia. (After O. Kirchner and H. Boltshauser.)

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Leptosphæria avenaria Web. The conidial stage of this fungus is known as *Septoria Avenæ* Frank. The disease (Speckled Blotch of

Oats) occurs in France, Germany, England, and America. It has been transmitted to several wild species of *Avena* such as *A. brevis*, *A. barbata*, *A. fatua*, *A. nuda*, *A. sterilis*, and *A. strigosa*, but not to other cultivated or wild *Gramineæ*. The ascigerous stage of the fungus is so far only known from artificial inoculations in the greenhouse (Weber, 1922).

Herpotrichia nigra R. Hart. This fungus invests whole twigs of spruce, mountain-pine, and juniper with a greyish-brown web of hyphæ. The mycelium kills the needles and twigs, often the whole tree. The needles are covered with a thick felt, they wither and become distorted but generally remain attached to the shoot. The perithecia, which appear in autumn as black dots in the mycelium on the under surface of the needles, are globose, somewhat flattened, and with long, brown appendages. The asci contain eight four-celled, colourless spores. The disease has been found in Denmark in closely planted stands of spruce where the trees were from ten to twenty years old. As a preventive measure it is recommended that trees should not be planted in situations where heavy drifts of snow are likely to occur.

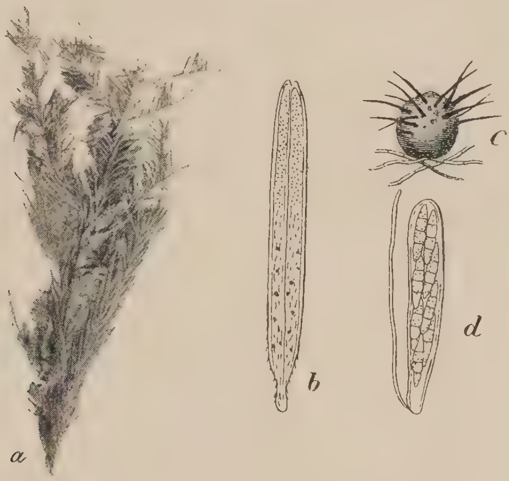

FIG. 286.—*Herpotrichia nigra*.

a, twig of spruce with web of mycelium; *b*, spruce needle with fructifications; *c*, single fructification; *d*, ascus and paraphysis. (*a*, after F. W. Neger; *b*–*d*, after E. Rostrup.)

White Root Rot of Vine

Rosellinia necatrix (Hart.) Berl.

This fungus, also known as *Dematophora necatrix* Hart., produces a network of mycelium in the cambium and wood of the root and of the base of the stem. The mycelium is composed of delicate, branched, transversely divided hyphæ, white to grey in colour and with here and there pyriform dilatations. On the surface of the infected roots there are found felted, greyish, rhizomorph-like strands of mycelium, from which branches arise and spread the fungus in the surrounding soil. Generally the mycelium remains sterile, but under favourable conditions, when the soil is moist, rows of small black sclerotia are formed on the surface of the roots. From these sclerotia, bundles of dark-coloured, branched conidiophores arise which abstrict ovoid, hyaline conidia. Within the sclerotia, on the dead roots, pycnidia containing large brown spores are produced and finally, after several years, perithecia develop upon the decayed roots. The perithecia arise on the outside of the sclerotia, between the conidiophores, and are globose with an apical papilla. The wall of the perithecium is black on the outside and white inside ; it bears asci together with paraphyses. The spores are fusiform and pass out of the ascus in the form of small, black drops.

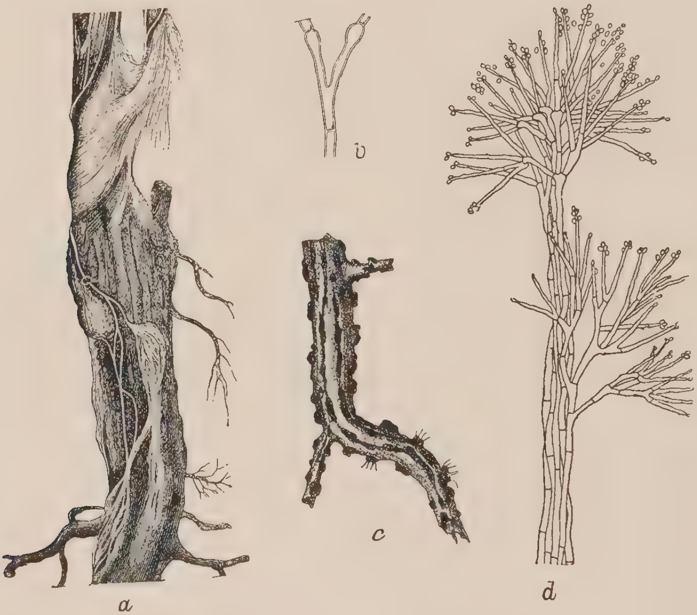

Fig. 287.—White Root Rot of Vine.
a, dead root ; *b*, bunch of mycelium ; *c*, portion of root with groups of sclerotia ; *d*, conidiophores growing from the sclerotia. (After P. Viala.)

This disease has been known from earliest times in the vine-growing countries of Europe—Austria, Hungary, Germany, France, Italy, etc., also in America and Japan, etc. It attacks a number of varieties of *Vitis vinifera*, *V. riparia*, *V. æstivalis*, *V. Coignetii*, and others. The fungus also lives on the roots of other kinds of trees such as plum, cherry, apricot, pear, apple, olive, oak, beech, maple, fir, and pine, etc. In rare cases it has also been observed on the roots of such plants as potato, pea, and mangel.

Control Measures :

(1) All infected plants, together with the roots, should be carefully dug up and burnt. (2) Ground from which infected vines have been removed should have a trench 1½–3 feet deep dug round it and the soil thrown up on the inside. The affected area should be sown with cereals or some similar crop for 5–6 years. (3) Special care must be taken to remove all infected roots before planting vines on ground where the disease has occurred. If any such roots are afterwards found, the soil should either be treated with carbon bisulphide (about 7 ozs. per sq. yd.) or left fallow for some years. (4) Manuring the vines with sulphate of iron (from 4–7 ozs. per vine) is recommended.

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Rosellinia quercina R. Hart. The fungus producing Root Rot of Oak not only attacks young, one- to ten-year-old plants, but also other young trees such as beech, maple, spruce, etc. The leaves from the top of the plant downwards become yellow and wither in consequence of the infection of the roots. The roots are invested with rhizomorph-like strands of mycelium, white at first, later brown. From these strands the mycelium spreads through the soil from plant to plant. The fructifications are found on the surface of the diseased roots and consist of conidia, which act as summer spores, sclerotia which are small, black structures of the size of a pin's head and serve for the over-wintering of the fungus, and lastly perithecia which are globose, black, with protruding beak and enclose eight unicellular, cymbiform ascospores. The inner tissues of the root (cortex and medulla) are permeated by the mycelium and killed. The blackened wood decays and passes into a condition of white rot. The outbreaks of disease are most severe in wet years in seed-beds. The disease was first detected in North-West Germany in 1880, and two years later it was found in Denmark (R. Hartig 1888, 1900).

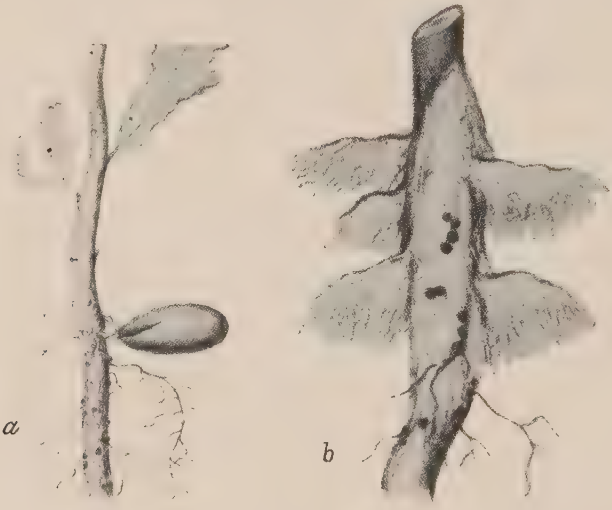

FIG. 288.—*Rosellinia quercina.*
a, root of two-year-old oak with sclerotia ; *b*, portion of root with aerial mycelium and small perithecia. (After E. Rostrup.)

Blight or Die-Back of Stone Fruits

Clasterosporium carpophilum (Lév.) Adhl.

This fungus, which is also named *Coryneum Beyerinckii* Oud. and *Stigmina Briosiana* Farn., produces roundish to semicircular spots on the leaves of peach, apricot, plum, cherry, almond, etc. The spots are distributed over the whole leaf, the centre part of a light colour, the margin dark. Fruits of all ages may be attacked by the fungus ; on young fruits the sunken spots are mostly of the size of a pin's head and have a red margin. The flesh of the fruit does not rot but assumes a corky appearance. The spots caused by the disease are brown to black, frequently covered by the stromata of the fungus, and, in the case of cherries, they may be so deep as to reach the stone of the fruit. Where the spots occur on the leaf, the tissue drops out, causing the leaf to appear to have been riddled by shot ; severely affected leaves fall prematurely. The spots on young twigs are elongated and brown ; there is often, too, an excretion of gum in the angles of the branches.

On the twigs and fruits there develop small stromata with exposed pustules of elongated, four- or multi-celled conidia (*Coryneum Beyerinckii* Oud.) and pycnidia which enclose pycnoconidia (*Phyllosticta Beyerinckii* Vuill.). It is not yet possible to say to what extent certain perithecia (*Ascospora Beyerinckii* Vuill.), which appear in spring, are genetically related to the fungus.

The mycelium of the fungus lives intracellularly in all the infected parts of the plant, growing through the cells crosswise. The hyphæ are sometimes slender, sometimes knotted and bent. The conidiophores always break through in tufts, sometimes so thickly on fruits and shoots that they form a real stroma. The conidia are oblong, divided by three, four, or more septa. When the conidia germinate they put out germ-tubes from several or all of the cells, either all at the same time or shortly after each other. Older conidia which have not germinated, as well as certain portions of mycelium occurring in diseased parts of the plant, divide up into single chlamydospores which may possibly assist in the over-wintering of the fungus.

There is always gummosis with this disease, and numerous inoculation experiments indicate that each wound arising in the period from June to November and extending to the cambium will be stimulated to produce gum if spores of the fungus enter.

The disease was first observed in 1889 in Southern Germany, and since the beginning of the present century it has spread to most European countries.

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Fabræa maculata (Lév.) Atk., also named *Endomosporium Mespili* (DC.) Sacc., sometimes causes much damage to pear stocks. In spring, the young leaves show minute, carmine-red spots which later become raised, black, and shining. The leaves are ultimately spotted all over with brownish-black spots and fall to the ground, the young trees within a short time being completely defoliated. Lesions also occur on the stem-parts of the tree in the form of elliptical, somewhat sunken spots. When the raised spore-masses on the leaves burst, the stroma which produces the conidia (*Morthiera Mespili* Fuck.) is exposed. Soon afterwards the growth of the stock ceases almost completely. The conidia are four-celled, each cell, except the basal one, carrying a bristle or flagella; they germinate easily in water. In December dark brown, usually single, perithecia are produced alongside the living conidial spore-masses on the diseased leaves. These perithecia, which ripen in April and May, contain asci, each ascus with eight hyaline, ovoid, two-celled spores which are arranged in two rows and between the spores are single paraphyses.

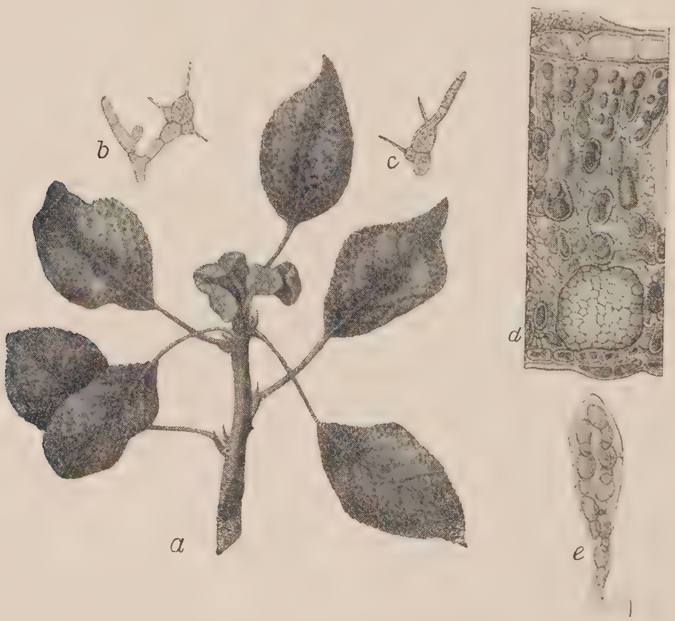

Fig. 289.—*Fabræa maculata.*

a, tip of pear stock with conidial-masses (*Morthiera Mespili*) on upper surface of leaf; *b* and *c*, conidia, germinated and ungerminated; *d*, beginning of perithecium; *e*, ascus. (*a–c*, Orig.; *d* and *e*, after P. Sorauer.)

The dangerous nature of the fungus was described in Germany in the seventies of last century (Sorauer, 1877; Lucas, 1879). In 1883–84 the fungus appeared in Sweden and in 1909 in America, causing much damage in both countries (Sorauer, 1886; Eriksson, 1886; Atkinson, 1909).

Fabræa Fragariæ (Sacc.) Kleb. produces irregular, reddish-brown spots on the leaves and leaf-stalks of the strawberry plant. On dead leaf-stalks which have been kept moist, brownish-white eruptions containing conidia can be observed (*Marssonia Fragariæ* Sacc.). The conidia are colourless and drawn out at the upper end to a short, fine point. After the fungus has over-wintered, perithecia are produced in the following spring (Klebahn, 1924).

In America (North Carolina) a similar disease is known on strawberry plants and sometimes causes considerable damage (Wolf, 1924).

Mycosphærella Fragariæ (Tul.) Lindau produces round, confluent, purple-red spots on the leaves of strawberries ; the spots soon dry up and the tissues below fall out. In summer there are subepidermal aggregations of mycelium which break through the epidermis and develop tufts of conidiophores (*Ramularia Tulasnii* Sacc.). In autumn, pycnidia are found (*Ascochyta Fragariæ* Lib.), and in winter sometimes also perithecia (Klebahn, 1918). Certain sorts of strawberries such as Progressive, Aroma, Gibson, and Charles I. are stated to be resistant (Gregory, 1925). In America, where the fungus has appeared very destructively in certain States (Illinois, Minnesota), Bordeaux mixture applied in spring when growth begins and again before flowering is recommended as a preventive. **M. sentina** (Fuck.) Schröt. gives rise to numerous circular, white spots with black margins on pear leaves. Conidia (*Septoria pyricola* Desm.) are formed in summer and autumn. The fungus sometimes produces premature defoliation of the trees (Osterwalder, 1922) ; it also attacks *Cydonia vulgaris*. Spraying the trees with 1½–2 per cent. Bordeaux mixture at the end of May and again 2–3 weeks later is advised (Klebahn, 1918). **M. cerasella** Aderh. produces round, brown spots, often with red margins, on cherry leaves. The spots, which develop tufts of conidia (*Cercosporella cerasella* Sacc.), soon die and drop out. Perithecia are met with in spring (Miyake, 1923). **M. Ribis** (Fuck.) Kleb. is found on the leaves of a number of species of *Ribes* such as *Ribes Grossularia*, *R. rubrum*, *R. nigrum*, and there gives rise to brown, sharply-defined spots on which septoria-tufts arise (*Septoria Grossulariæ* [Leb.] West. et al.). Perithecia, approximately globose in shape, are formed later in the leaf tissue. **M. Mori** (Fuck.) Lindau produces irregular, light brown spots, with a darker margin on the leaves of the mulberry tree. A subepidermal conidial cushion (*Cylindrosporium Mori* Berl.) is present in the spots, and in winter, perithecia are found on the fallen leaves. This disease has been observed in Hungary. **M. Fraxini** (Niessl.) Lindau appears on the leaves of *Fraxinus Ornus* in Southern Italy. In the stomatal cavities of the leaves a knot of mycelium is produced from which conidiophores (*Cercospora Fraxini* [DC.] Sacc.) are developed.

Later, pycnidia (*Phyllosticta fraxinicola* Curr.) and perithecia appear (Scalia, 1909). **M. castanicola** (Desm.) Berl. causes the leaves and fruits of the chestnut (*Castanea vesca*) to wither and dry up. Minute, angular, reddish-brown spots appear on the leaves which curl spirally, become dry, and drop in wet weather. The fungus often gives rise to red, later black, spots on the fruits. Three types of fructifications have been recorded : filiform, bent conidia (*Cylindrosporium castanicolum* [Desm.] Berl.), pycnidia (*Phyllosticta maculiformis* [Pers.] Schröt) and perithecia. This fungus was found in France in 1847 ; it was first recorded as a dangerous pest in Mid-Italy in 1893 and led to the complete destruction of the crop in some places (Berlese, 1893). **M. Hippocastani** Kleb. gives rise to small, roundish, reddish-brown, sharply-defined spots on the leaves of the horse chestnut (*Æsculus Hippocastanum*). In severe attacks the whole leaf becomes brown and withered. Tufts of conidia (*Septoria æsculicola* [Fr.] Fuck.) appear on the leaf-spots and after hibernation the perithecia, single or in small groups, develop. The fungus has made its appearance each year since 1904 near Cuxhaven (Klebahn, 1918). **M. laricina** R. Hart. causes the shedding of the needles of larch. In June and July the needles show brown spots on which minute, black, conidial cushions are present ; perithecia appear in the following spring on the fallen needles.

Black Rot of Grapes

Guignardia Bidwellii (Ell.) Viala & Rav.

This disease appears in June on the leaves of the vine as brown, scattered spots which have well-defined margins. These spots soon dry up and a little later, in July, when the grapes are about two-thirds of the full size, or perhaps earlier, reddish-brown spots appear on the berries. In damp, hot, misty weather the spots on the grapes may spread until the whole berry is covered. After a few days the spots on the leaves, and soon afterwards those on the grapes as well, show minute, globose, reddish-brown to greyish-black pycnidia which are embedded in the tissues. The spores in the pycnidia—the pycnoconidia—are extruded in a tendril-like form through an opening at the top of the pycnidium ; they germinate at once and spread the disease during the growing period. These pycnidia were formerly regarded as a separate species of fungus—*Phoma uvicola* B. & C. Together with the pycnidia are found sunken spermogonia (formerly named *Nœmaspora ampelicida* Eng.) which develop rod-shaped spermatia. After the production of pycnoconidia ceases in late autumn, the cavity of the pycnidium is filled with a dense, homogeneous mass of mycelium which in the course of the winter develops into a sclerotium; in spring the whole of this changes to a perithecium. By means of the spores which are formed in the asci of the perithecium the fungus hibernates and continues its work of destruction in the following year.

If at the end of the growing period the conditions are unfavourable to the normal development of the fungus, *e.g.* continued cold and dry weather at harvest-time and unsuitable supply of plant food, then there is a formation of chlamydospores in the infected grapes. Certain of the mycelial hyphæ widen out and become divided by septa into thick-walled cells which are very resistant to changes in weather conditions.

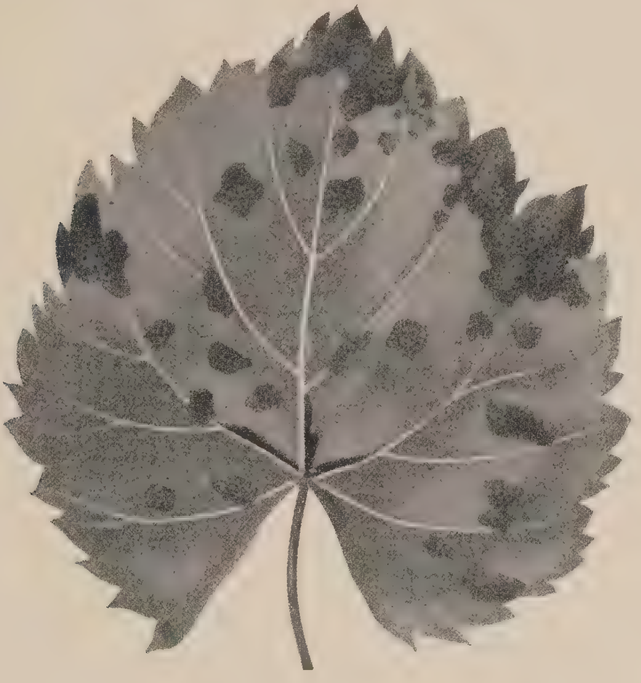

Fig. 290.—Black Rot of Grapes. (After P. Viala.)

The vine itself is not attacked by the fungus, but the effect of the disease on the berries is often such as to completely destroy the crop in a few weeks.

This disease has been known since 1848 in America and since 1885 in France, where it has spread extensively; it has also appeared in Italy.

Control Measures:

(1) All infected grapes should be gathered and burnt. (2) All the dead and diseased parts of the vine should be removed in spring, and in

June and July, from the time the buds begin to swell, spraying with Bordeaux mixture (2–3 per cent.) should be practised several times.

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The **Caucasian Black Rot,** *Guignardia baccæ* (Cav.) Jacz. is closely related to the last-mentioned disease; it has been observed in the Caucasus, South of France, Italy, Algeria, and Asia Minor. This form attacks the berries, rarely the leaves. The perithecia appear in autumn between the pycnidia and they have an elongated neck. The asci are longer than those of *G. Bidwellii*. The pycnoconidia are at first fusiform (*Phoma flaccida* Viala) and later slightly curved (*Ph. reniformis* Viala & Rav.). This form appears to be endemic in Europe (v. Speschnew, 1899; Delacroix and Maublanc, 1909).

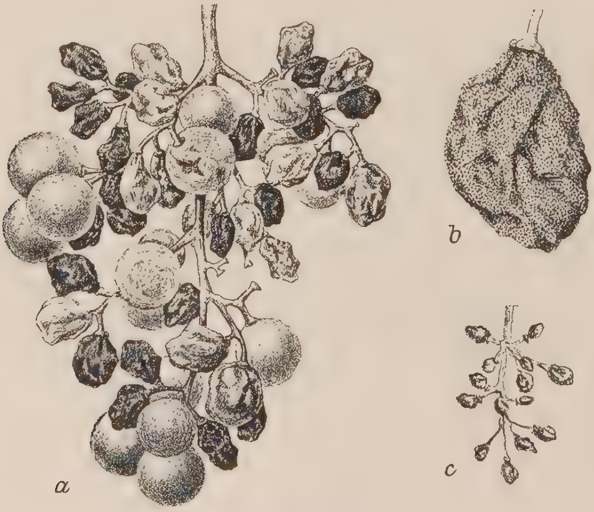

Fig. 291.—Black Rot of Grapes.

a, bunch of grapes, some berries fully developed, others killed; *b*, a diseased berry (enlarged); *c*, a group of diseased berries. (After E. Prillieux.)

Apple Scab or Black Spot of Apple

Venturia inæqualis (Cook.) Aderh.

Fusicladium dendriticum (Wallr.) Fuck.

This disease appears in two forms, one on the leaf and the other on the fruit. The leaf form is characterized by black spots on the surface of the leaves and to a less extent on the leaf-stalks ; more rarely it occurs on the young branches and shoots. In the case of young trees the leaf spots may be of considerable size, as big as a hazel nut or even larger, and are sharply defined. On older trees the spots may spread until the whole of the surface of the leaf is covered. The black colour of the spots is due to the conidiophores and conidia ; the white colour of the periphery of the spots is due to the leaf tissue becoming separated and the space filled with air. In the epidermal cells the hyphæ follow the outer wall as a stroma and put out, here and there, a tuft on which conidiophores and conidia develop. The conidia are pear-shaped, generally one-celled, sometimes two- or many-celled. The disease is spread by the conidia and apparently also by the conidiophores, even by isolated stroma-cells. As soon as the conidiophores are old they cease to produce conidia, and if detached the terminal cell germinates, the

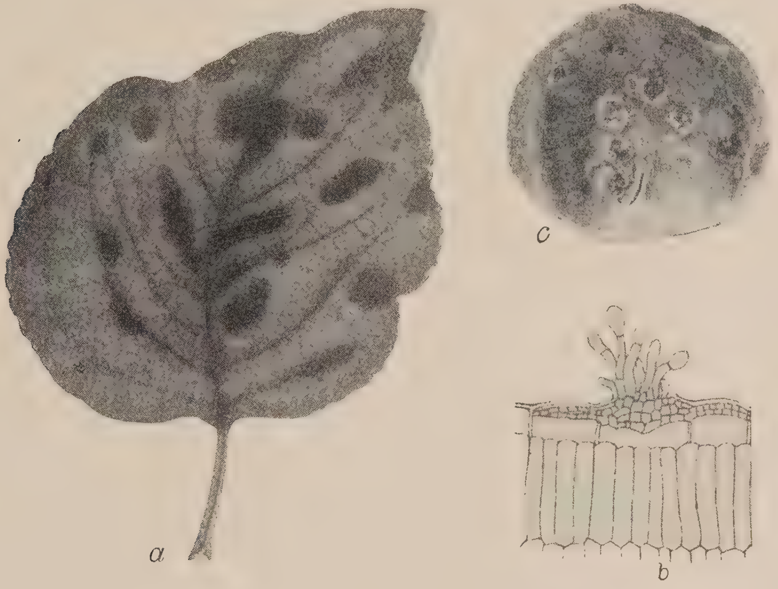

Fig. 292.—Apple Scab.

a, apple leaf attacked by *Fusicladium dendriticum ;* *b,* apple attacked by scab ; *c,* cross-section of a scab spot on leaf. (Orig.)

germ-tube reaches a considerable length and becomes divided into a number of segments which round off and separate themselves from one another.

This form of the disease on the leaf was first described in 1833, but it was not until the 'seventies that it appeared in European orchards (Sorauer, 1874, 1875; Rostrup, 1879). It caused much damage in Sweden in 1882 and 1884. The leaves turned black and fell from the trees, large numbers lying on the ground in the middle of summer. The small crops which were common in these years were, no doubt, largely due to this disease.

The form of the disease on the fruit used formerly not to be as injurious as it has become of late. The first appearance of scab on the fruit is similar to that on the leaf. The epidermal cells in which the mycelial stroma lies are forced apart and the stroma is laid bare. In the cell tissues of the flesh underlying the stroma, a layer of cork is formed and separates the diseased tissue from the healthy. Generally, conidia are not produced in the diseased spots, but the individual stroma cells multiply and spread the infection.

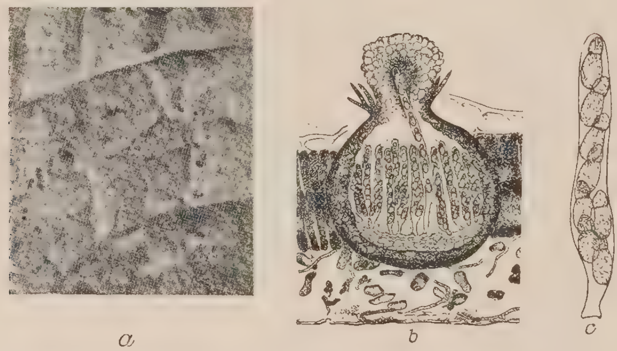

Fig. 293.—Apple Scab.
a, portion of leaf with perithecia; *b*, a perithecium; *c*, an ascus. (After E. S. Salmon and W. M. Ware.)

The infected apple leaves fall to the ground and in the course of the winter the next stage in the life-history of the fungus develops. In February to March minute, black warts, which are the openings of perithecia embedded in the leaf tissue, can be found on the decayed leaves. The asci in the perithecia contain each eight spores. Inoculations made with these spores result in the production of fully-developed conidial pustules by the middle of May. It is in this way that the early outbreaks of disease occur. Cold, wet weather in spring favours the development of the fungus, whereas warm, dry conditions check it.

There can be no doubt that apple scab is one of the most serious diseases met with in fruit cultivation. The growth of the shoots is disturbed and the tips die back, the supply of food material to the tree is hindered, and the production of fruit diminished. The fruit itself is stunted and falls prematurely. Lastly, scabby apples can only be sold with difficulty.

Investigations lately carried out in England leave no doubt that the outbreaks of disease in spring are mainly due to the ascospores which are produced in the perithecia on dead leaves. On certain varieties such as Cox's Orange Pippin, Worcester Pearmain, and Bismarck, the disease appears at an earlier date than on Newton Wonder, Beauty of Bath, Bramley's Seedling, Lane's Prince Albert, and Annie Elizabeth. Thus the first-named varieties can serve as centres of infection if early spraying with a fungicide is not practised. Pre-blossom spraying is, therefore, very important with these varieties, so that the mass-production of infective summer spores may be prevented and a severe outbreak of disease avoided.

Control Measures :

(1) As far as possible the fallen leaves from diseased trees should be collected and burnt. (2) In the following spring the trees should be sprayed with 2 per cent. Bordeaux mixture before the buds reach the pink stage, a further spraying with 1 per cent. Bordeaux mixture after blossoming, and again with the same strength of fungicide 2–3 weeks later.

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Pear Scab

Venturia pirina Aderh.

Fusicladium pirinum (Lip.) Fuck.

Pear scab is found on the leaves, shoots, and fruits. Black, scab-patches appear on the leaves, sometimes on both surfaces, sometimes only on the under surface. In the case of very susceptible varieties the fungus may develop to such an extent that after a period of three years a branch may be almost covered with a continuous layer of scab-wounds, or it may have been killed. On the fruit the fungus first produces black, circular spots about the size of a pea, and these spots extend in all directions until the greater part of the fruit is covered. At the same time, cracks arise in the fruit in various directions, crossing one another and penetrating deeply into the flesh. The mycelium only spreads in the outer layers of the cells of the diseased portions. The conidiophores, in places, have a wavy appearance but are mainly erect. The conidia are pointed at both ends; they germinate readily in water.

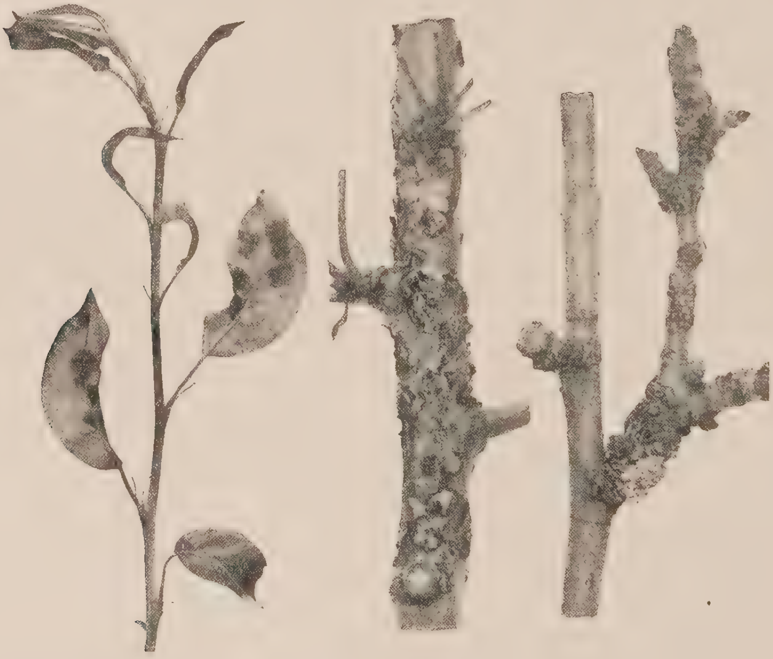

Fig. 294.—Pear Scab. Left, a one-year-old shoot, attacked by scab. Middle, a three-year-old shoot badly attacked. Right, a shoot, part of which is living and the other part dead. (Orig.)

The form of fungus on the leaf was observed and described in 1840 in

France; in 1864 the fungus was found on pears in Bohemia. The disease was further studied in Germany in 1874 and in France in 1877 and 1878.

Just as in the case of apple scab, the perithecia develop on the dead leaves during winter; they contain asci each with eight spores which ripen in March or April and are then ejected from the ascus in large numbers. The spores at once infect the young shoots and fructifications which are being formed at this period. How far a similar formation of perithecia also takes place in the peripheral parts of the infected one- or two-year-old shoots has not been investigated.

In years when scab is prevalent it is found that the different varieties of pears vary considerably in their susceptibility. It has been observed, for example, in Sweden, that some sorts such as Flemish Beauty, Jakob's Birne, and Larsmässebirne are often so cracked and deformed as to be inedible.

As the over-wintering organs of the fungus are not only present in the dead leaves lying on the ground but also, in the case of certain especially susceptible varieties, in considerable number on the young twigs, the control of the disease is more difficult than in the case of apples.

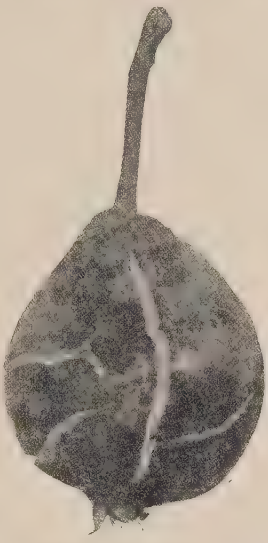

FIG. 295.—Pear Scab. Deep cracks produced on the fruit. (Orig.)

Control Measures:

(1) In late autumn all shoots attacked by the scab fungus should be cut out and burnt, the older branches and stems should be painted with 2–3 per cent. Bordeaux mixture, and all fallen leaves should be collected as soon as possible and burnt. (2) In the following spring the trees of susceptible varieties should be sprayed with 2 per cent. Bordeaux mixture at the time when the blossoms open, then with 1 per cent. Bordeaux mixture immediately after the blossom is over, and a further spraying, also with 1 per cent. Bordeaux mixture, 2–3 weeks later. In the case of espalier trees any valuable dessert fruit should be enclosed in paper bags so as to protect it against rain and subsequent infection.

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The following are other forms of *Venturia* found on trees and shrubs : **Venturia Cratægi** Aderh. on the fruits and leaves of species of white thorn. The perithecia develop on the over-wintered leaves (Aderhold, 1913.) **V. Tremulæ** (Frank) Aderh. on varieties of poplar. Large, irregular, brownish spots with black margins, often covered with a delicate, olive-green felt, occur on the upper surface of the leaves of the young shoots. From these spots conidia are produced. The leaves curl, wither, and fall. In the next spring, perithecia with bristles show themselves on the under surface of leaves lying on the ground (Laubert, 1923). **V. Aucupariæ** Lask. (Rostr.) on the leaves of young mountain ash trees. Reddish-brown, sharply limited spots with conidiophores and conidia on the upper surface of the leaf. Pycnidia with fusiform conidia and perithecia with yellowish-green, two-celled spores. **V. Fraxini** (Rostr.) Aderh. on the leaves of the ash. Single patches of conidia are found in summer. The leaves wither at an early stage, curl up, and fall. In the following April the perithecia, which bear black bristles, become ripe. The disease has sometimes been the cause of much damage in nurseries and plantations of young ash trees. **V. chlorospora** (Ces.) Aderh. on species of willow. Patches of conidia, of an almost ink-black colour, arise on the leaves and young shoots. In the following spring, scattered groups of perithecia occur on the dead leaves lying on the ground. **V. Rhododendri** Tengw. on the leaves of *Rhododendron ponticum*. Perithecia with long, stiff bristles at the ostiolum are found on the under surface of the leaf. The conidial stage of the fungus is *Phyllosticta Maximi* Ell. & Everh.

In this connection may also be mentioned : **Fusicladium Cerasi** (Rabh.) Erikss., although the perithecial stage (*Venturia*), which possibly belongs thereto, has not yet been observed. Cherry scab forms small, round, sometimes depressed spots on the fruits, more rarely on the leaves of sweet cherries. The disease prevents the ripening of the fruit and causes it to fall when only half ripe.

Didymella Lycopersici Kleb. attacks the stems of tomato plants and causes the parts above the point of attack to rapidly die. Young stems are often diseased at the ground level and fall over. The conidia are extruded as long, thin, brownish tendrils from the pycnidia which arise on the infected parts of the stem. On diseased stems that have wintered in the open the pycnoconidia develop in large numbers; when the stems are kept in a damp situation for some time perithecia are formed between the pycnidia. This fungus was the cause of heavy losses in 1919 near to Hamburg; it has also been found in Holland, England, and Denmark (Schœvers, 1919; Klebahn, 1921).

Didymosphæria populina Vuill. is regarded in France (Prillieux, 1897) as the original cause of the dying of the pyramid poplar in Mid-Europe. At first only the young terminal shoots dry up, but later thick branches also. In the dead portions of the bark, pycnidia with colourless, elliptical conidia develop, and in autumn, perithecia with brown, two-celled spores. Conidial pustules of *Napicladium Tremulæ* (Frank) Sacc. are also found on the leaves; the conidia are multicellular. In Denmark (Rostrup, 1902), another fungus *Dothiorella sphæroides* Pers. is regarded as the cause of this disease of poplars. Finally, it has been suggested that the main causes of the dying back of the twigs are the continuous raising of trees from cuttings and a succession of years in which the weather conditions are unfavourable.

White Rot of Grapes.

Charrinia Diplodiella (Speg.) Viala & Rav.

Coniothyrium Diplodiella (Speg.) Sacc.

The disease first appears, as a rule, on the pedicels, which turn brown and shrivel. From the pedicels the fungus spreads to the berries, which acquire a lead-grey colour and dry up with an infolding of the skin. Greyish-white, pustular pycnidia arise on the surface of the infected parts. The pycnidia are developed from a stroma of interwoven hyphæ which is laid bare by the rupture of the cuticle. Under the grey, wart-like excrescence, a round cavity—a pycnidium—is formed and from its base pycnoconidia arise. After pushing off the cap of mycelium the conidia pass out through a small opening at the apex; they are ovoid, colourless at first, brown later, and they are able to infect the vine through wounds on the epidermis. A high temperature in summer and moist atmospheric conditions favour the spread of the disease. The conidia remain viable until the following summer. When the pedicels and berries have dried up, the whole bunch of grapes generally drops off. In certain cases "mummied" fruits are produced which remain attached to the twigs and develop pycnidia. The conidia which arise from these pycnidia spread the disease.

The fungus passes from the stalk of the grape to the young shoots, spreads round them and develops a swelling above the ring; such a shoot generally dies back. Sometimes the fungus passes to the woody parts of the vine, in which case the bark peels off and the wood is exposed.

In addition to this stage of the fungus, which has been named *Coniothyrium Diplodiella*, a perithecial stage has also been observed. The perithecia are formed in October and November on the pedicels; paraphyses are found together with the asci.

The fungus was first reported from Italy in 1878 and was then described. In 1886 it caused serious damage in the Vendée and in 1887 in Herault; it is also known in Austria-Hungary, Belgium, and America.

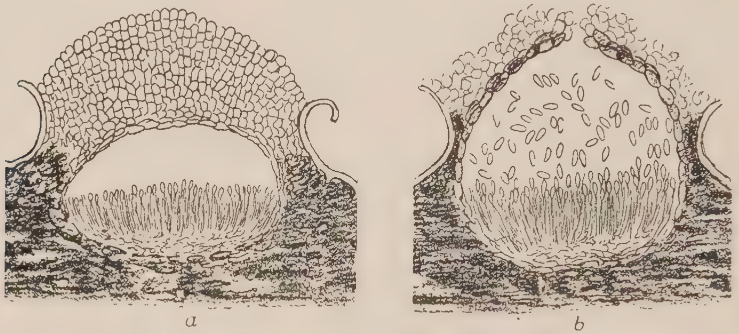

Fig. 296.—White Rot of Grapes.

a, subepidermal pycnidium in section; *b*, pycnidium with raised apical pore. (After E. Prillieux.)

Control Measures:

(1) All infected parts of the vine should be cut out, down to the healthy wood, and burnt. (2) The vines when cut back should be sprayed with petroleum and set alight. (3) When all diseased parts of the vine have been destroyed the soil should be soaked with petroleum and set alight. (4) The vines should be sprayed repeatedly with 4 per cent. Bordeaux mixture; the mycelial cap which covers the opening of the pycnidium acts like a sponge and assists the fungicide to enter the cavity of the pycnidium.

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Cherry Leaf Scorch

Gnomonia erythrostoma (Pers.) Auersw.

This disease is found on the young leaves of sweet cherries, more rarely on the sour varieties. Shortly after the leaves unfold in spring a few large, pale spots appear and later a considerable number of small, brown ones. Pycnidia arise on the under side of the spots in July and August and about the same time perithecia begin to develop within the leaf. The diseased leaves become stunted and the petioles bend over. The pycnoconidia are rod-shaped and are usually bent at one end into a hook. The cherries remain small and stunted and fall to the ground whilst still unripe.

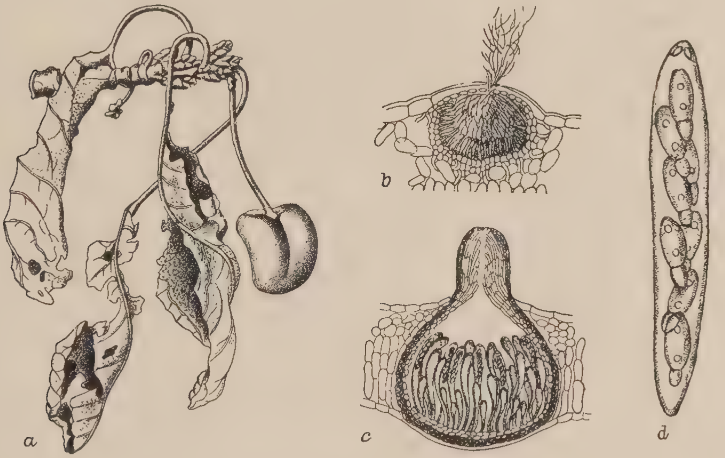

Fig. 297.—Cherry Leaf Scorch.
a, diseased leaves and cherries ; *b,* spermogonium in section ; *c,* perithecium in section ; *d,* ascus. (After A. B. Frank.)

The diseased leaves with their bent leaf-stalks remain on the tree throughout the winter, while the healthy ones fall in the normal manner in autumn and rot in the course of the winter. The parts of the leaves which still remain on the tree persist sufficiently long to allow of the ripening of the perithecia ; this occurs in April and May. The perithecia are globose with a long neck, the ascospores two-celled, the lower cell only half as big as the upper one ; they germinate at once and infect the new leaves which are being put out.

This fungus was the cause, for several years, of much damage to sweet cherries in Altenland near Hamburg, the crop being almost entirely destroyed. Trees that suffer repeatedly from outbreaks of the disease become stunted and finally die. Latterly the disease has been very prevalent in Baden and Switzerland.

As the fungus lives and hibernates in the leaf and leaf-stalk only, it would be possible by collecting and destroying all leaves remaining on the tree in autumn to prevent the infection of the new leaves in the following spring.

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Gnomonia padicola (Lib.) Kleb. occurs on the leaves of *Prunus Padus* and produces radiate, brown patches of mycelium which grow rapidly and give rise to a tree-like branching of the hyphæ. The mycelium appears to be entirely superficial, but it is in reality covered by the cuticular layer of epidermal cells. In addition to the surface mycelium there are hyphæ which penetrate between the cells of the mesophyll and obtain therefrom nourishment for the fungus. From the surface strands of mycelium the conidial receptacles are developed and they finally rupture the cuticle and become open pustules. The conidia are elongated, colourless, unicellular ; they are not dispersed as dust but they separate from one another when water reaches the leaves. The conidial stage of the fungus is generally named *Asteroma Padi* DC.

Within the tissues of diseased, over-wintered leaves the perithecia develop. They have the form of a flattened ellipsoid with a long beak-like neck the tip of which rises above the under surface of the leaf. The asci are linear and eight-spored, the spores thread-like, divided across the middle and provided at each end with a gelatinous prolongation (Klebahn, 1908).

Gnomonia veneta (Sacc.) Kleb. In the conidial stage (*Glœosporium nervisequum* Fuck.) the fungus forms large, brown areas along the main nerves of the leaf lamina of the plane (*Platanus vulgaris*) and also on the young twigs. On the under surface small brown pustules arise. The conidia are liberated from the pycnidia in the form of worm-like tendrils. Sometimes the fungus lives perennially in the twigs, and the young leaves become infected whilst still in the buds. The conidial form *Glœosporium Platani* Mont., which occurs on twigs of the plane tree, seems to belong together with this fungus. **G. leptostyla** Fr. gives rise to spots on the leaves and green fruits of the walnut (*Juglans regia*). The spots appear in July and August, some are large, others small, grey with a brown margin; those on the fruits coalesce to such an extent that they cover the whole surface. On these spots, tufts of conidiophores are produced; the conidia are two-celled and bent into a sickle-shape.

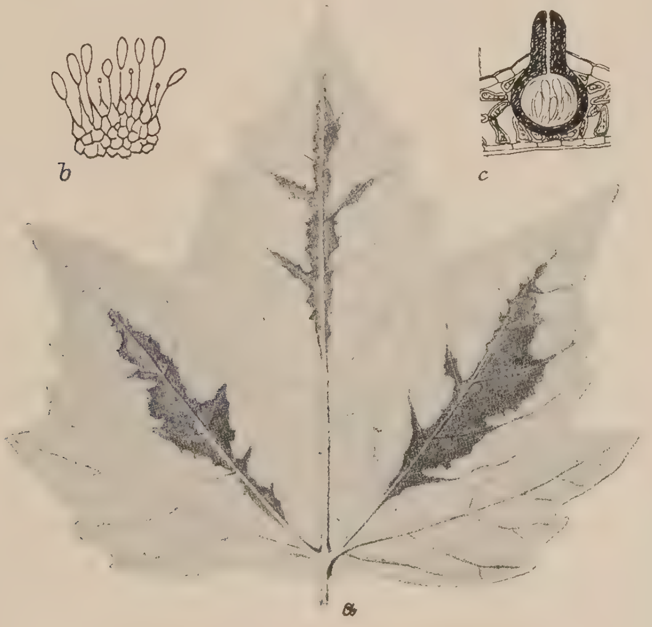

FIG. 298.—*Gnomonia veneta.*

a, plane leaf with patches along the main veins; *b*, conidial stage; *c*, perithecium. (*a*, after E. Rostrup; *b* and *c*, after F. W. Neger.)

In this stage the fungus has been named *Marssonia Juglandis* Lib. On the withered leaves lying on the ground there develop in the following spring, reddish-brown perithecia which contain fusiform, two-celled ascospores. Infected fruits fall to the ground before they are ripe. It is recommended to collect and destroy the diseased fruits and leaves as a means of controlling the disease.

A closely related fungus, **Plectodiscella veneta** Burkh., the conidial stage of which is named *Gleosporium venetum* Speg., occurs on the twigs of *Rubus occidentalis*, *R. Idæus* var. *aculeatissimus* and *R. neglectus* and has been reported from America (Burkholder, 1917).

Gnomonia Disease, or Anthracnose, of Lime

Gnomonia Tiliæ (Oud.) Kleb.

This disease appears on the leaves of the lime (*Tilia grandifolia*, *T. ulmifolia*, etc.) in May. The spots occur either singly or in numbers crowded together; they are round, clear, and sharply limited and the leaf tissue at these points is killed right through and dried up. Often the spots are found at the edges of the leaves but are generally spread over the whole of the surface. Minute black dots are found on the upper side of the larger spots. Similar spots also occur on the leaf-stalks with the result that the stalk dies and breaks, causing the leaf to fall. By the middle of summer, diseased trees are largely defoliated and the ground below is covered with fallen leaves, some of which are still green. Blackish, sunken patches also occur on the lower part of the annual shoots.

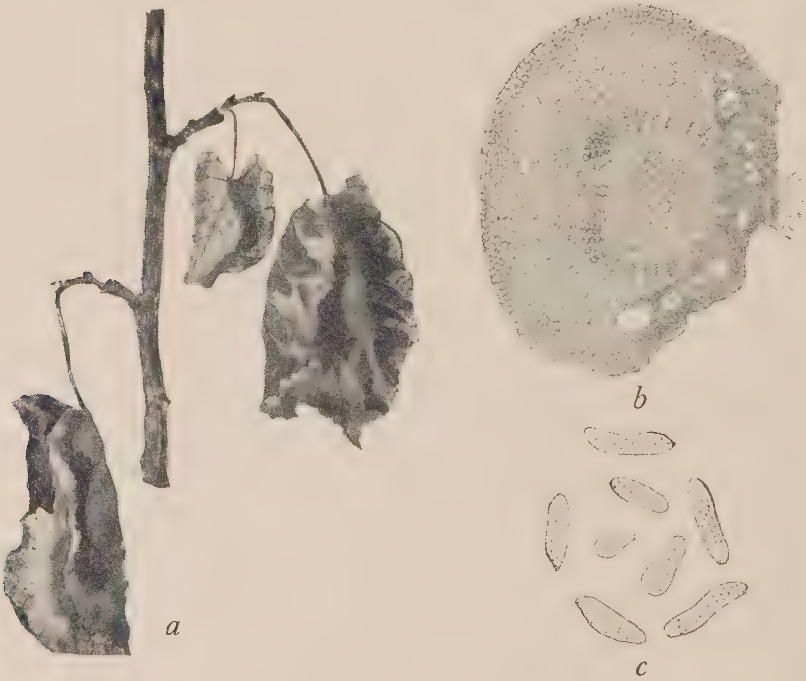

FIG. 299.—Gnomonia Disease of Lime.
a, diseased leaves and part of stem of annual shoot; *b*, cross-section of stem through a conidial pustule; *c*, conidia. (*a*, Orig.; *b* and *c*, after R. Laubert.)

A cross-section of the leaf-stalk or twig shows the conidial pustules, which are at first covered by a layer of epidermal tissue but later become exposed by the rupture of the covering. Short, colourless conidiophores arise, each of which abstricts a conidium at its tip; the conidia are ovate and somewhat constricted in the middle. This stage of development of the fungus is named *Glœosporium Tiliæ* Laub.

A small number of perithecia (each with a beak directed downwards) occur between the smallest veins of the over-wintered leaves which are usually much decayed. The ascospores are two-celled, the septum being near the lower end.

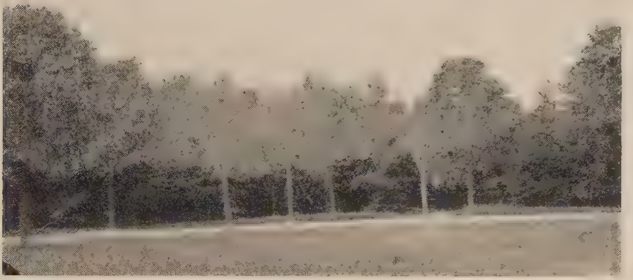

Fig. 300.—Lime Trees in the Castle Park, Malmö, attacked by the Gnomonia Disease and almost defoliated. (Orig.)

This fungus was first observed and described in Holland in 1883 (Oudemans). In the 'eighties it was found on *Tilia grandifolia* and *T. ulmifolia* in Holland, Austria, and Italy, as well as on *T. parvifolia* in Germany. In 1902 the fungus was noted in Denmark and in 1904 and 1918 in Germany, where lime trees in avenues were much damaged. A serious outbreak occurred in the Castle Park at Malmö (South Sweden) in 1924, against which spraying was carried out in the following spring but without favourable results.

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Valsa leucostoma (Pers.) Sacc. attacks cherry trees and causes large branches and whole trees to die. The foliage becomes yellow and withered. Gummosis occurs in fissures of the bark. Chambered pycnidia (*Cytospora leucostoma* Aderh.) and perithecia occur together in the stroma. The ascospores are one-celled, hyaline, usually slightly curved. The fungus can only enter by wounds or cracks and is not able to penetrate healthy twigs ; under certain conditions it gives rise to canker-like growths (Aderhold, 1903, 1905 ; Spieckermann, 1906). In America, two strains of the fungus have been distinguished, the one producing both conidia and ascospores, the other conidia only (Leonian, 1923). **V. Prunastri** (Pers.) For. on plum, peach, and apricot trees. The bark and cambium in the affected parts become yellow and rotten. The pycnidial stage was earlier named *Cytospora rubescens* Fuck. **V. oxystoma** Rehm on *Alnus viridis.* The mycelium blocks the vessels and hinders the flow of sap. The perithecia, which ripen on dead twigs, are long and bear some bristles. In the case of 15- to 20-year-old trees the fungus can cause a "die-back" of the tips of the twigs (Appel, 1904). **V. spinosa** Pers. has been observed in Denmark to cause the bark of the stem and twigs of 10- to 30-year-old birches to split lengthwise and to lead to the dying-back of the twigs.

Chestnut Bark Disease

Endothia parasitica (Murr.) Clint.

This disease is found in the United States on *Castanea dentata* and *C. sativa*, more rarely on *C. crenata*, *C. pumila*, and *C. alnifolia ;* a number of species of *Quercus* are also liable to it. Brown, raised spots appear in spring on the young shoots while long cankers, which tend to

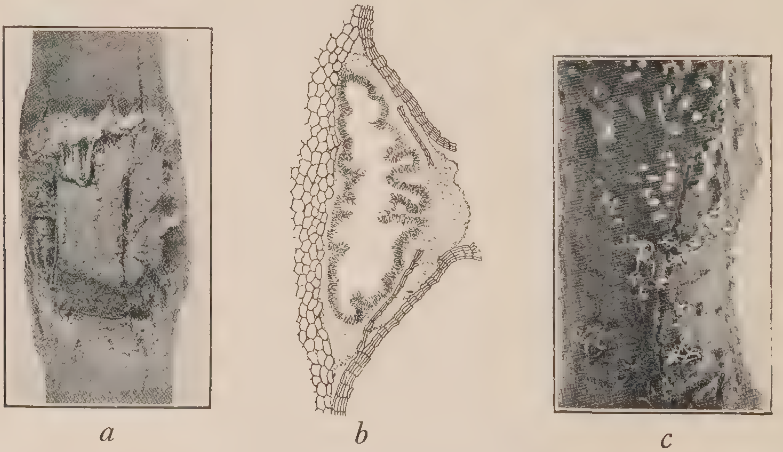

Fig. 301.—Chestnut Bark Disease on *Castanea dentata*.
a, cankers on vigorous shoot ; *b*, vertical section of pycnidial pustule ; *c*, pycnidial pustules with "spore-horns" developed in wet weather. (*a*, after C. L. Shear, N. E. Stevens, and R. J. Tiller ; *b*, after F. D. Heald ; *c*, after H. Metcalf and J. F. Collins.)

girdle the branch, are formed on the older twigs. In the course of the summer, the leaves on the infected branches lose their colour, turn yellow, and wither. Orange-yellow to reddish-brown fructifications develop on the affected parts. These fructifications are of two kinds—brownish-yellow, multi-chambered pycnidia from which, in damp weather, long tendrils of pycnoconidia are discharged, and black-spotted perithecia which contain asci, each ascus with eight two-celled ascospores. Trees attacked by the fungus are ultimately killed. The disease is caused by the fungus *Endothia parasitica* (Murr.) Clint., which is also named *Diaporthe parasitica* Murr.

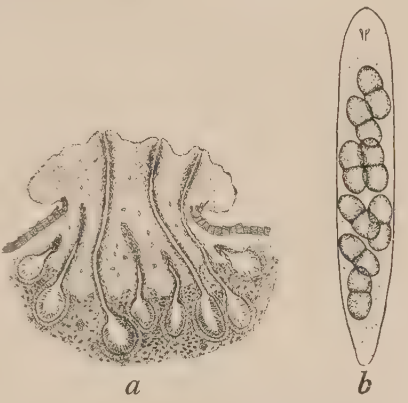

FIG. 302.—Chestnut Bark Disease on *Castanea dentata*.

a, vertical section of a perithecial pustule, three of the perithecia cut so as to show the full length of the necks; *b*, ascus with eight spores. (After F. D. Heald.)

This disease was first observed in 1904 in New York State, having been introduced in a consignment of nursery plants from China. Since its introduction it has spread to a number of the other States in America and has caused much damage; the losses even in the year 1911 were estimated at about 25,000,000 dollars.

In 1926 the disease was stated to have completely laid waste the chestnut stands north of the Potomac River and east of the Alleghany Mountains, while

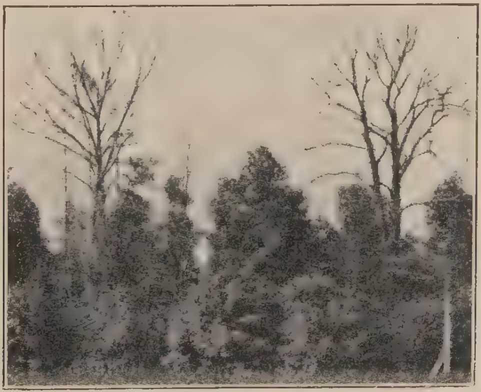

FIG. 303.—Large Chestnut Trees killed by the Bark Disease. (After H. Metcalf and J. F. Collins.)

the forests of the Southern Appalachians are apparently doomed to destruction. A county by county survey begun in 1924 has revealed infections at the southern limit of the commercial chestnut-growing region, and it is estimated that by 1930 more than half and by 1935 nine-tenths of the Southern Appalachian counties, covering an area of 30,000,000 acres, will show over 80 per cent. of infection in their chestnut stands (Gravatt and Marshall, 1926). In Canada the disease made its appearance in 1923, in the Niagara Peninsula and in Norfolk County. In the last-named county 25 per cent. of the trees were diseased and it is regarded as probable that "the disease will not be checked until all the chestnut timber is gone" (Stone, 1923).

In Europe (Belgium) the disease has been once observed (Metcalf, 1924) but has not been met with again (Marchal, 1925). By a decree of March 8, 1921, the importation of living plants, fruits, and seeds of this chestnut into France has been prohibited.

Recently other species of chestnut have been imported into America and there tested as substitutes for *Castanea dentata* which suffers so badly from the disease. The varieties that are being tried are *C. sativa* from Europe, *C. mollissima* (more than 10,000 seedlings have been distributed in the United States during the past ten years), *C. Henryi* and *C. sequinii* from China, *C. crenata* from Japan, and others (B. T. Galloway, 1926). It is not yet possible to state whether these efforts will meet with success.

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Ink Disease of Chestnut

Melanconis perniciosa Briosi & Farn.

This is the most dangerous of the fungous diseases that attack the European edible chestnut. It is found on the parts of the plant above ground as well as in the root system. The first sign of the disease is that the male catkins fail to flower and fall undeveloped to the ground, whilst the foliage, which has abnormally small leaves, turns yellow in July. The rudimentary fruits are small and the seed capsule which bursts before the fruit is ripe remains attached to the tree. The bark of the stem dries up and comes away from the wood which is laid bare. Sometimes a black, inky fluid is exuded at the base of the stem. In the second year of the disease the affected twigs die back and in 2–3 years the tree itself is killed, although on heavy soils some trees may live for 4–5 years.

The disease makes its first appearance in the root by causing the fine branches to become black and brittle, and from this point the infection spreads centripetally to the large root-branches.

For the past fifty years or so this disease, which was first discovered in the North of Italy, has been the subject of much investigation. The opinions which are held as to the cause and development of the disease, as well as to the conditions which are favourable to an outbreak, are still marked by considerable divergencies. On the one hand it is considered that infection takes place through the parts of the tree above ground, the spores of the fungus entering by way of the lenticels or cracks in the bark. The mycelium arising from this infection passes through the cambium ring of the larger branches and main stem to the root, quickly reaching the tips (Briosi and Farneti, 1907–11). The name given to the fungus is *Melanconis perniciosa* Briosi & Farn., in the conidial stage *Coryneum perniciosum* Briosi & Farn., and in the pycnidial stage *Fusicoccum perniciosum* Briosi & Farn. On the other hand, the origin of the disease is considered to be in the roots (Petri, 1912–14; Dufrénoy, 1922, 1923), the infected sap rising from the centres of decay through the stem and branches into the young shoots and leaves. The organisms responsible for the decay are thought to be either various forms of fungi and bacteria such as *Torula exitiosa* Sey. (Seynes, 1879), *Armillaria mellea* Vahl. (Planchon, 1882), *Diplodia Castaneæ* Sacc. (Gibelli, 1883), *Mycelophagus Castaneæ* Mang. (Mangin, 1903), *Blepharospora cambivora* Petri (Petri, 1924), etc., or unfavourable soil conditions, such as lack of humus (Delacroix, 1897) or a shortage of plant food in the soil (Pestana, 1907), etc. These factors have, it is claimed, a weakening effect upon the mycorrhizal fungi which are concerned in nutrition or on the root fibres themselves.

This disease, which can appear on types of soil varying from those which are rich in humus to those which are almost sterile, is found in all countries where chestnuts are grown.

Control Measures :

(1) In the early stages of the disease the tree should be pruned, as this reduces the size of the head and by establishing a better proportion between head and root enables the tree to grow away from the disease. (2) Badly infected trees should be removed and burnt. (3) Towards the end of November the soil surrounding the tree should be watered with a 20 per cent. solution of sulphate of iron, and again at the end of

February. (4) European varieties of chestnut might be grafted on some species of *Quercus*, such as *Quercus pedunculata*, *Q. palustris*, *Q. sessiliflora*, *Q. acuminata*, *Q. ambigua*, etc., or on *Castanea crenata* from Japan.

In all the grafting experiments which have been made the best results have come from Japanese stocks. Trials carried out in France and extending over twenty years have shown the grafted Japanese trees to be resistant while the European trees were attacked by the disease.

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Closely related to the above fungus is **Melanconis Modonia** Tul. which also attacks the chestnut and produces similar lesions on the twigs. Its conidial stage, *Coryneum modonium* (Tul.) Griff. & Maubl. appears as small brownish-black pycnidia on the bark.

CHAPTER XV

DISCOMYCETES

THE body of the fungus, which is termed an apothecium, may be fleshy or waxy, in the form of a cup, basin, or disk. It is usually raised from the substratum on a stalk, which may be long or short. On the upper face of the fructification lie numerous asci arranged side by side and mingled with narrow paraphyses.

Larch Canker

Dasyscypha Willkommii R. Hart.

This fungus attacks the twigs, branches, and stems of the larch tree (*Larix decidua*). The disease occurs on trees of all ages, from the two-year-old seedlings in the nursery to the oldest trees in the forest, the most injury being caused to trees of 10–30 years of age. The fungus gains entrance to the tree through injuries caused by frost, hail, breakages due to snow or wind, insects, etc.

The first sign of the disease is the yellowing of the needles on some of the shoots or on the head of the tree, and the needles wither in a half-developed state. Below the yellow tuft of needles there is generally a considerable exudation of sticky resin from an abnormally thickened portion of the bark. On older branches the disease often appears on one side only in the form of small or large, dead, sunken, blackish cankers. These cankers, in the middle of which dead stumps of branches are often situated, can go on increasing in size and depth for several years. The mycelium spreads through the various tissues of the branch or stem, resulting in an active production of turpentine and a browning of the tissues. In late autumn and spring the growth of the fungus reaches its maximum and comes to a standstill in summer. Sometimes the diseased twigs with their bunches of needles wither in the spring; in fact, the whole tree may die within a year. In other cases, fresh bunches of needles may develop and the tree may continue to live for some years before being ultimately killed.

On the cankers, as well as on the adjacent bark, small yellowish-white pycnidia arise and produce one-celled, hyaline conidia. Later, snow-white pustules break out and develop into apothecia which are dish-shaped and have a short stalk. The outside of the apothecium has a white, down-like coating and inside is a bright orange-red hymenium. The apothecia require about a year for their full development and ripening; they open and close according to the humidity of the atmosphere. The hymenium consists of a layer of asci mingled with paraphyses; the asci contain each eight ellipsoid, hyaline ascospores which are forcibly expelled into the air in late autumn.

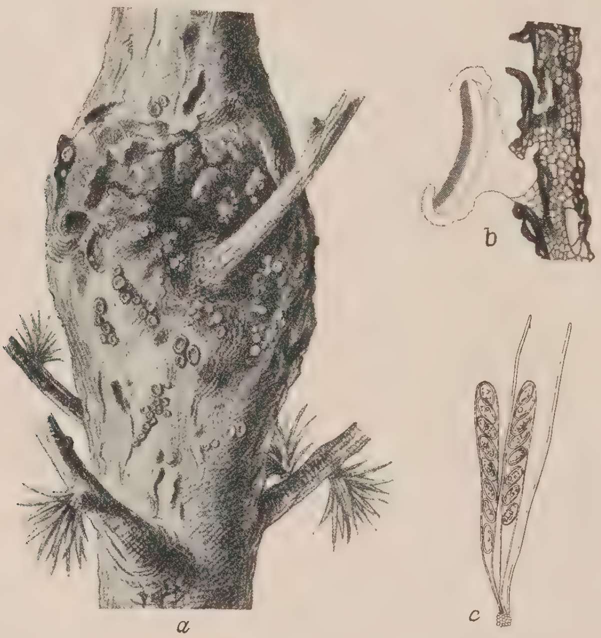

Fig. 304.—Larch Canker.

a, stem of larch with cankers and dish-shaped apothecia; *b*, cross-section of portion of bark bearing apothecia; *c*, asci and paraphyses. (*a*, after E. Rostrup; *b* and *c*, after M. Willkomm.)

The fungus is endemic in the high Alps and it causes the greatest havoc in valleys and situations where the air is stagnant and the soil wet; in many places it has put an end to the growing of larch. The disease appears to be prevalent throughout Northern and Mid-Europe where the larch is grown. The fungus has also been found on *Larix sibirica*, *L. Dahurica*, and *L. americana*.

The control measures which are recommended are: (1) To grow mixed stands of trees. (2) To avoid low and moist situations. (3) To carefully remove all infected trees or parts of trees.

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Dasyscypha fuscosanguinea Rehm. attacks badly-grown pine trees, generally on the main stem about one-third from the top ; the food supply is cut off, and the upper part of the tree dies. Some trees perish almost at once, others live on for several years. In the latter case, cankers, which are covered with resin, develop in the course of a year or two. On the dead parts of the bark, the apothecia break out, either singly or in groups ; they are jug-shaped in the early stage, with a short stalk, brownish-grey without, and covered with brown hairs and scales. When ripe the dish-shaped hymenium is rust-yellow to bright orange-red. The asci contain each eight ellipsoid, one-celled, hyaline spores. This fungus has been found in Tyrol on *Pinus pumilia* and *P. Cembra*. In Northern Europe (Norway, Northern Sweden, Finland) it often causes much loss (Lagerberg, 1912). **D. calyciformis** Willd. is usually saprophytic on twigs of a number of conifers, sometimes parasitic on *Abies sibirica* (Zürich), *A. pectinata*, and *Pinus silvestris* (Saxon Switzerland). The hymenium of the apothecium is pure yellow.

Clover Rot

Sclerotinia Trifoliorum Eriks.

This disease often appears in autumn on young clover plants growing from seed sown in spring corn. Brown spots arise on some of the leaves and spread rapidly and soon afterwards the leaf and stem begin to wilt. Generally these symptoms do not attract the attention of the farmer, but in the following spring the sickly condition of the crop is very noticeable as many of the plants are wilted and their leaves and stems lie collapsed on the ground. Often there are patches of varying size in which all the plants are dead and in severe attacks almost all the clover is killed. Black, irregularly-shaped sclerotia are found loosely attached to the neck of the root of the dead plants.

The fungus causes the most injury to red clover, but it has been found also on alsike clover, white clover, and crimson clover, as well as on *Onobrychis sativa*, *Medicago lupulina*. *M. sativa*, and many other closely related species, even on *Vicia Faba*. In the case of red clover, the sclerotia attain the size of a pea or somewhat larger and develop on the surface of the root. On alsike clover the sclerotia are much smaller and may be present also on the above-ground parts of the plant even up to the tips of the shoots. In the case of lucerne the upper part of the root decays and the sclerotia surround the root neck. Not infrequently the strongest of the lucerne plants recover, thanks to their deep rooting system. Above the dead part of the plants, buds arise in the course of the summer and develop shoots which grow above the surface of the ground.

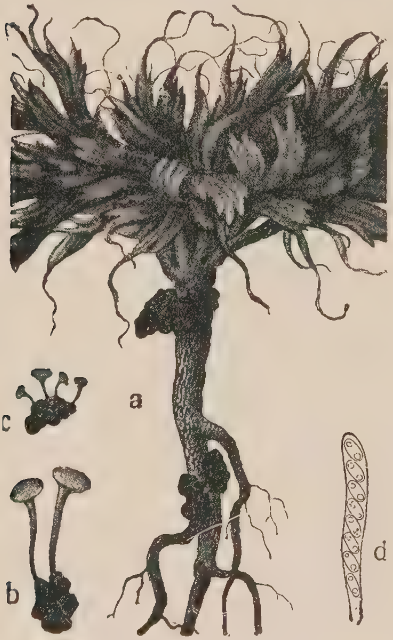

Fig. 305.—Clover Rot.

a, clover plant killed by fungus, with sclerotia on roots; *b* and *c*, sclerotia from which fructifications have grown out; *d*, ascus. (Orig.)

In late autumn, apothecia grow out of the sclerotia; they are pale red in colour and are borne on long, narrow stalks which are often irregularly bent and twisted. The length of the stalk varies according to the depth of the germinating sclerotia in the soil, the stalk serving to raise the disk-shaped, patulous portion of the fructification above the ground. The asci, each of which contains eight spores, lie close together on the upper surface of the disk. The ascospores are able to germinate at once and give rise to a mycelium which penetrates into the young plant and spreads within the tissues to such a degree that, finally, only the epidermis and vascular bundles remain, the rest of the tissues having been displaced by the mycelium.

In wet weather, white flocculent coatings of mycelium appear on the surface of the plant, particularly on the neck of the root, and from them sclerotia are formed. The sclerotia can live for a long time in the soil and may even become quite dry without losing their viability. The infection seems, as a rule, to be limited to young clover plants which explains why the fungus appears mainly in the early spring of the year following the sowing, whereas two-year-old and older crops are rarely attacked. Mild winters, wet weather, and close sowing favour the development of the disease.

The susceptibility of the clover appears to be influenced by the origin of the seed. In Bavaria in 1911 the clover crops grown from seed obtained from Eastern, Western, and Southern Europe suffered much more severely from the disease than did those grown from Mid-European seed. It is not impossible that the disease is also carried in the seed.

Control Measures :

(1) If in spring only a few plants are found to be diseased they should be dug out and burnt, together with any sclerotia that can be found; the bare patch of ground can be sown with grass seed. (2) If a large number of plants are attacked, the field should be ploughed up and some other suitable green crop sown. (3) Infected ground should be rested from clover for three or four years. (4) If a reappearance of the disease is feared, clover should only be grown in grass mixtures. (5) As apparently the disease can be spread by the seed, efforts should be made to obtain the seed from a healthy crop.

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Sclerotinia Nicotianæ Oud. & Kon. gives rise to whitish spots, which are formed of interwoven hyphæ, on the stems and leaves of the tobacco plant. On the mycelium, small branches are formed which, at their tips, develop hyaline, spherical conidia in short chains. From these spots, black sclerotia are produced which fall to the ground, and towards the end of the winter give rise to stalked apothecia if the soil be wet. To protect the tobacco plants from this disease they may be surrounded by a crop of *Phaseolus vulgaris* (but not *Ph. multiflorus*), which throws off its leaves before the tobacco harvest, and reduces the humidity of the atmosphere between the tobacco plants (Oudemans and Koning, 1902, 1903).

Sclerotinia Rot

Sclerotinia sclerotiorum (Lib.) Fuck.

Some of the species of *Sclerotinia* appear on the underground parts of plants. An example was seen in 1913 in the neighbourhood of Stockholm, where some of the plants in a crop of Jerusalem artichokes, which had grown normally to a height of about 6 feet, suddenly changed in

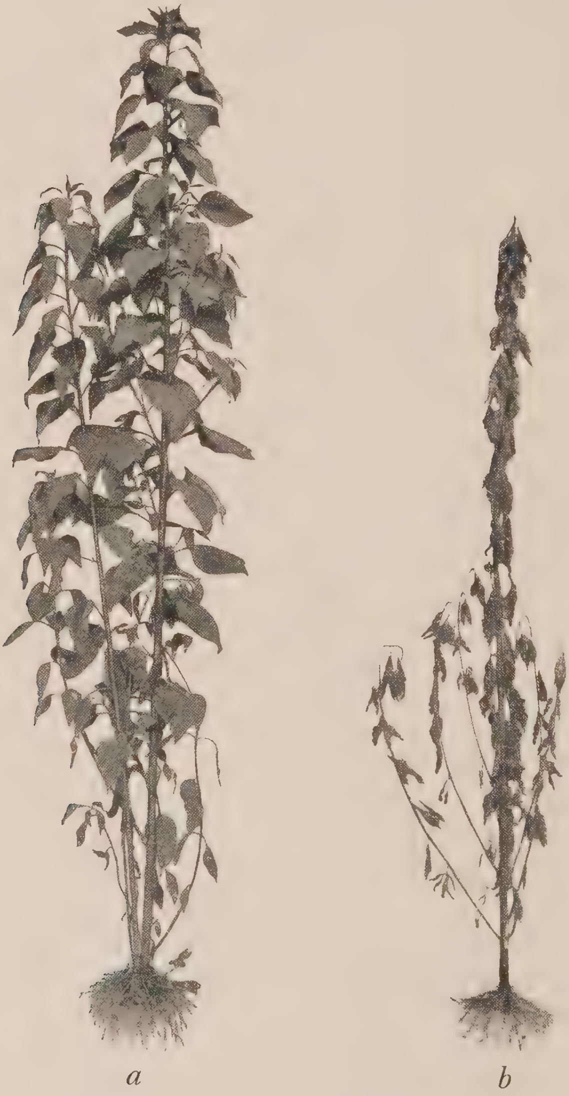

Fig. 306.—Two Jerusalem Artichoke Plants.
a, healthy; *b*, killed by Sclerotinia Rot. (Orig.)

appearance. The leaves became flaccid and blackened and hung down, the plant ceased to grow, and on examination no artichoke tubers were found in the soil, only tumour-like masses of mycelium. On the surface of the dead stems, white pustules were formed, and within the stem, in the cavities, numerous sclerotia which were black on the outside and white inside. Similar sclerotia were also found on the artichokes formed by apparently healthy plants growing beside the diseased ones.

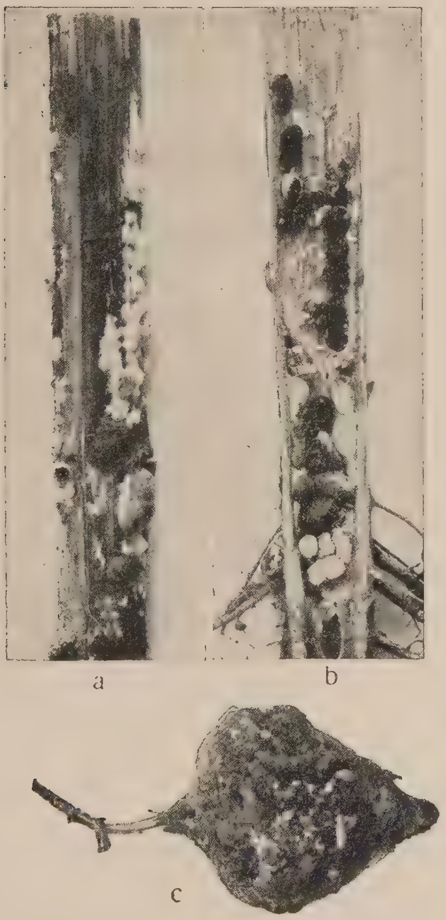

Fig. 307.—Sclerotinia Rot on Jerusalem Artichokes. *a*, portion of stem with white, cottony, fungus pustules on outside; *b*, section of stem showing sclerotia within; *c*, artichoke covered with sclerotia. (Orig.)

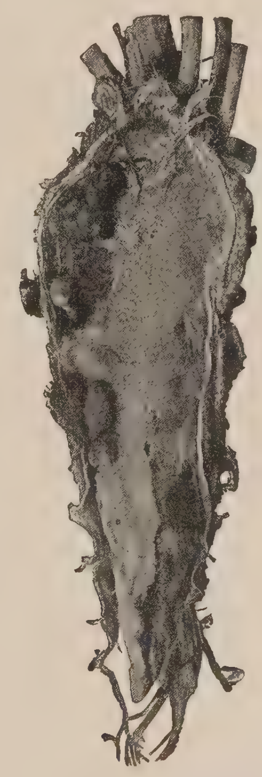

Fig. 308.—Sclerotinia Rot on Carrot. Large numbers of sclerotia inside the root. (Orig.)

There is a similar production of wart-like pustules and sclerotia, especially within the root, on carrots suffering from the same disease.

A form of disease which deserves special notice is one which occurs in Ireland on potatoes and is known as " Stalk Disease," " White Spot," " Falling at the Butt," etc. It appears at the beginning of July on the outside of the stem, generally on the lower part. Small, white, cottony warts are produced from which silvery drops of a water-clear fluid exude. These warts become denser and develop into hard sclerotia of a waxy consistency. Similar sclerotia also occur in the cavities of the stems. Shortly after the appearance of the disease, the stem becomes brittle at the points where it was first attacked and falls over to the side. The sclerotia on the outside of the stem are easily loosened and fall to the ground. When the stem breaks off or rots in the autumn the sclerotia in the cavities are freed. The sclerotia germinate in the following summer and can then spread the infective material to new potato plants.

Numerous other agricultural and horticultural plants, such as species of the genera *Brassica*, *Sinapis*, *Phaseolus*, *Carum*, etc., can be attacked by this fungus whilst they are still growing in the ground. The lesions appear as ashy or brownish-grey mould on the stems, leaves, flowers, and even fruits. The organs that are attacked are retarded in their development, and in the case of severely diseased plants there may be little or no formation of flowers and fruit.

The disease causes very severe damage to some crops : beet, mangels, turnips, kohl-rabi, scorzonera, celeriac, chicory, etc., when stored, and it can be recognised by the white, cottony felt of hyphæ and the luxuriant mycelium which spreads over the surface of the roots and also over stems and leaves when such are present. The growth and spread of the mycelium are favoured by moist air and the disease develops to a greater extent in the cellar than in the clamp. The disease spreads rapidly from root to root by means of the mycelium, which can continue to live as such from year to year on the floor of the cellar and in the cracks on the walls and ceilings ; this accounts for the return of the disease once it has appeared in a cellar. The fungus gradually penetrates into the stored roots or vegetables and brings about a decay of the tissues.

After a period, hard, black sclerotia appear in the white mycelial felt. The sclerotia vary in size from a pea to a large bean, they are often very numerous and packed closely together, their shape depends partly upon the substratum. On thick roots and in the cavities of stems and roots, the sclerotia are round and broad with numerous, irregular indentations ; on leaf-stalks and in the veins of leaves they are flattened and elongated. When the fungus is prevented by adverse external conditions from developing further at the normal time, it can remain alive for years in a resting condition by means of the sclerotia.

Fruiting bodies, apothecia, develop in the spring from the sclerotia, generally several from each sclerotium. The apothecia are from 1–3 cm. long and carried on a stalk through which runs a narrow canal. There is a depression in the middle of the disk which extends to the canal in the stalk and the apothecium has thus a trumpet-like appearance. The upper face of the disk is composed of closely-crowded asci, each ascus containing eight ellipsoid spores. When the spores germinate a mycelium arises which develops into a cottony felt on a suitable substratum.

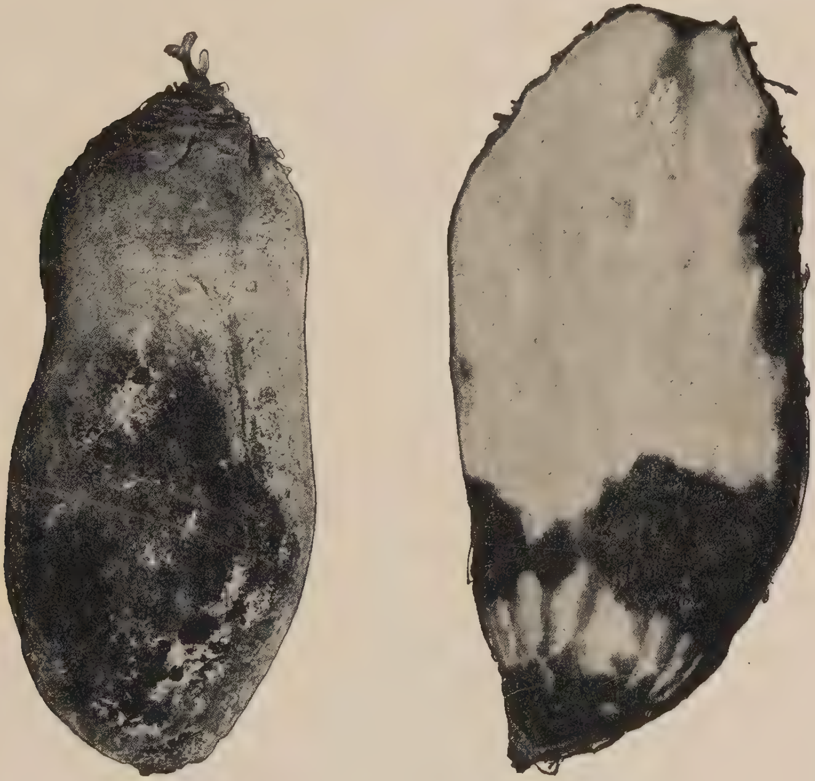

FIG. 309.—Sclerotinia Rot on Mangels.

a, mangel with numerous sclerotia on lower part of root ; *b*, section of mangel showing the spread of the rot within the root. (Orig.)

Control Measures :

(1) When harvesting the crop, any roots that are diseased or arouse suspicion should not be stored. (2) Any sclerotia that are found at harvest-time should be collected and burnt. (3) If a crop is to be stored in a cellar or building where the disease has appeared previously, the floors, walls, and ceiling should be well cleaned, or, better, the building

should be fumigated by burning sulphur. (4) When cellars are used as storage places they should be ventilated, the roots examined from time to time, and diseased ones removed. (5) Roots stored in clamps should first be covered with a layer of soil, then a layer of dry straw, and finally earthed over. (6) Roots that are slightly diseased should be cooked before being fed to stock. (7) Infected ground should be rested for three years from any crop that is susceptible to the disease.

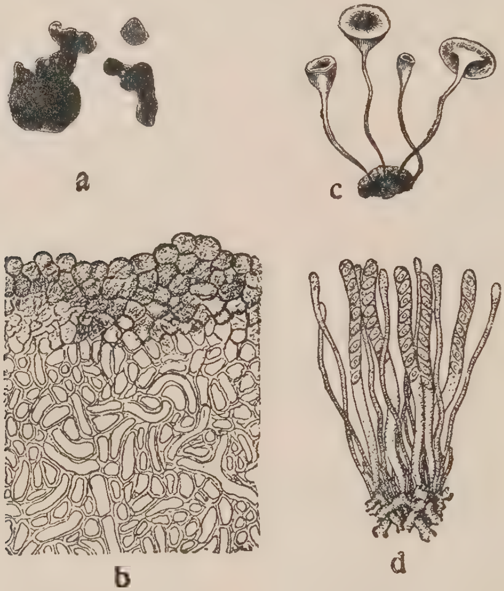

Fig. 310.—*Sclerotinia sclerotiorum.*

a, sclerotia; *b*, outer wall of divided sclerotium; *c*, sclerotium with four fructifications; *d*, asci and paraphyses. (*a* and *d*, after R. E. Smith; *b* and *c*, after E. Prillieux.)

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Brown Rot of Apples and Pears

Sclerotinia fructigena Schröt.

Monilia fructigena Pers.

This disease is recognisable immediately after the flowering period by the sudden and rapid wilting of some of the blossoms and leaves of the fruit spurs on apple, or pear, trees. The blossoms and leaves that are attacked hang down, brown and withered, but between the dead

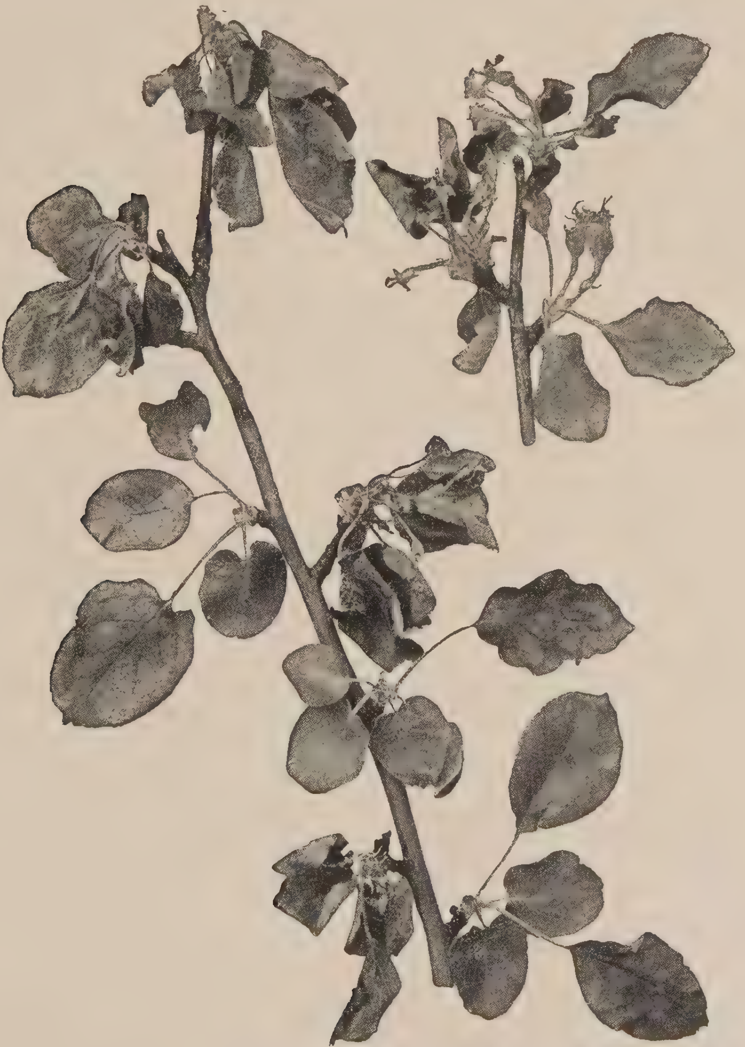

FIG. 311.—Brown Rot of Apple. Twig with blossom- and leaf-buds killed by the Monilia stage of the fungus. (Orig.)

rosettes of leaves healthy ones are also present. The fungus spreads to the bark of the branches adjacent to the diseased rosettes. In this stage the disease is termed " Blossom and Twig Wilt."

A week or two later, grey, wart-like pustules appear on the dead pedicels and leaf-stalks. The pustules are about the size of a pin's head and are covered with long chains of conidia ; the conidia are able to germinate at once. This stage of the development of the fungus can be described as " summer Monilia."

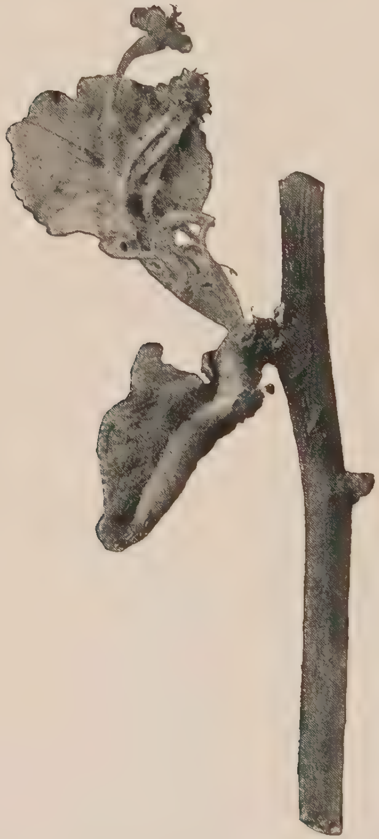

FIG. 312.—Piece of twig of apple showing dead, canker-like portions of bark. (Orig.)

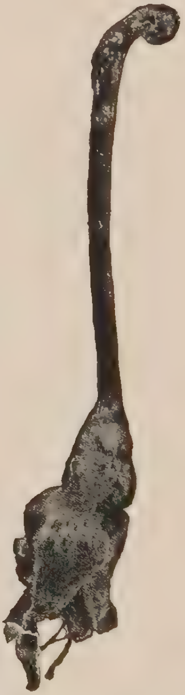

FIG. 313.—Apple flower killed by the Monilia stage of the fungus. Conidial pustules on the stalk. (Orig.)

In the following spring (March to April) whitish-grey conidial pustules, which have ripened in the course of the previous winter, appear on the dead, brownish-black parts of the branches killed by the summer attack ; this stage is the " previous season's Monilia."

There can be no doubt that this very early conidial generation is the main source of infection of the young blossoms and leaves which appear a few weeks later. The pustules contain an inexhaustible supply of conidia which are able to infect the tender flowers and leaves as soon as the buds open. The conidia are carried by the wind, or by insects, to the stigmas of the open flowers and there germinate. The germ-tube passes down through the style to the ovary and from there into the pedicel; as a consequence the flower and its stalk wither and die. From the flower stalk the fungus grows further into the twig on which the flower is carried and causes it to die gradually.

In addition to this "Monilia cycle" there is also the "Sclerotinia cycle" in which a stroma and apothecia are developed. This generation is seen in late summer on ripening apples and pears, especially on early varieties, in the form of "autumn Monilia." The diseased fruits begin to rot and numerous concentrically-arranged, grey pustules arise on the decayed portions; the annular arrangement of the pustules is regarded as being due to the variation of light by day and night (Molz, 1906). Sometimes, instead of rotting in the manner just described, the fruits are changed into black, sclerotia-like "mummies" (Black Rot). The rotting, greyish-coloured fruit usually falls to the ground, but the hard, black mummies, which are probably infected by the fungus *Monilia cinerea*, often remain on the tree, sometimes hanging there for a whole year or even longer. It has been possible to artificially inoculate one side of an apple with *Monilia fructigena* (concentric conidial pustules) and the other side with *Monilia cinerea* (hard, black mummies).

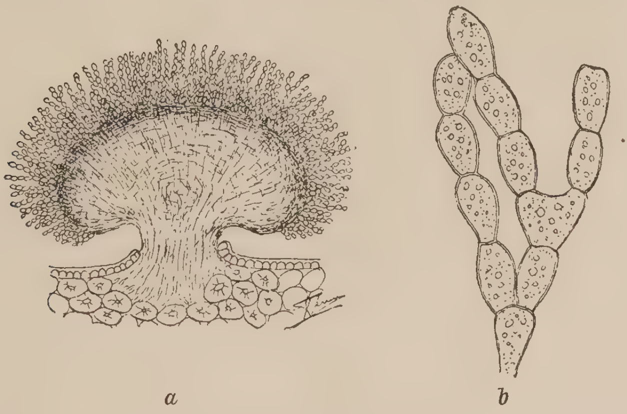

Fig. 314.—Brown Rot of Apple.
a, conidial pustule in section; *b*, chains of conidia. (After T. Ferraris.)

After over-wintering for two years in the open, a sclerotial stage of the fungus, with apothecia, is produced on the mummied fruits. The apothecia have a short stalk and a clay-coloured to greyish disk; the disk is at first funnel-shaped, then cup-shaped, and finally flat. The inner face carries asci with ascospores which can germinate at once. These spores are able to cause an outbreak of the "previous season's Monilia."

The different varieties of apples vary in their susceptibility to the disease. In England Lord Derby, Cox's Orange Pippin, James Grieve, and Ecklinville Seedling suffer severely (Wormald, 1917). In Sweden Weisser Astrakan, Hampus, and Oranie have shown themselves to be especially susceptible.

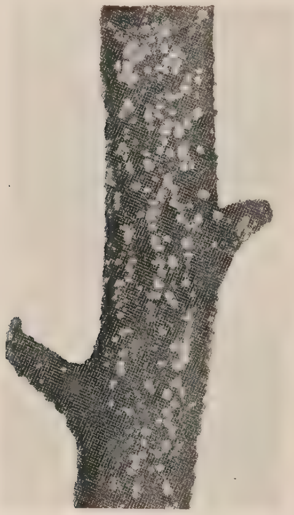

Fig. 315.—Branch of Apple Tree attacked in previous year by Monilia Wilt, in spring (April) covered with grey conidial pustules on the dried-up portions of the bark. (After H. Wormald.)

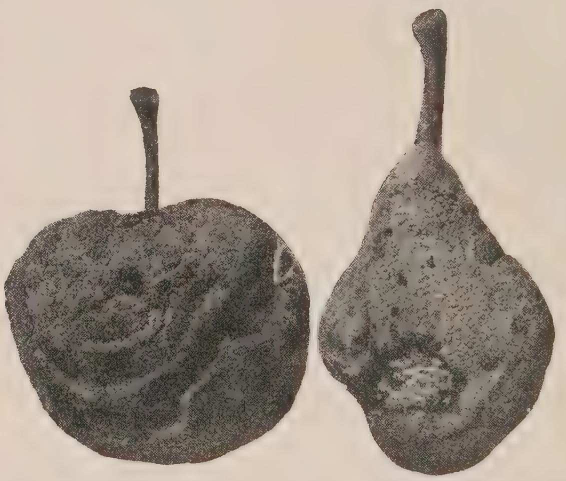

Fig. 316.—Brown Rot. Apple and pear bearing conidial pustules of the fungus. (Orig.)

The fungus can be transmitted to peaches, apricots, hazel nuts, and grapes (Sorauer, 1899, 1900).

The "autumn Monilia" form of the fungus with decayed or mummied fruits has been known since the beginning of last century. The "spring Monilia" form with blossom and twig wilt appeared much later. It is recorded in 1889 that this form of the disease had long been known in Tyrol (v. Thuemen), and there are definite accounts of its appearance in Europe in 1887 and 1889. Since 1894 the disease has been known as a very dangerous pest of fruit in practically all European countries.

Control Measures:

(1) Early in spring, before the winter buds have opened, an examination should be made of trees which have suffered in the previous year

from blossom and twig blight. If any of the old, diseased blossoms or twigs are found they should be removed and burnt, as well as the adjacent twigs, although the latter may appear healthy. (2) The tree should then be sprayed with 2 per cent. Bordeaux mixture. (3) If, in spite of these measures, dead blossoms, fruit spurs, or twigs are seen two or three weeks after the tree has flowered they must be cut off at once and burnt. (4) A second spraying with 2 per cent. Bordeaux mixture should be given in June. (5) In autumn, all decayed or mummied fruits, whether on the ground or still on the trees, should be collected and burnt.

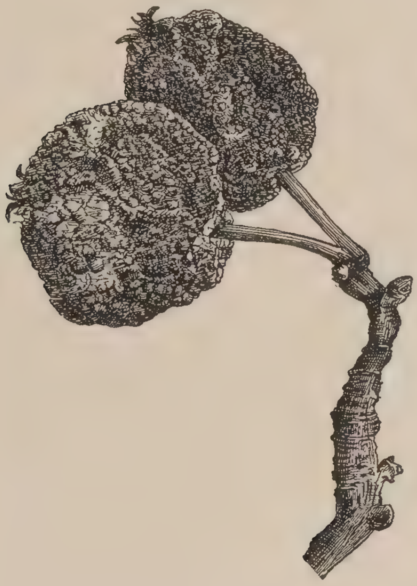

Fig. 317.—Brown Rot. Apple mummies that have remained on the tree over the winter. (Orig.)

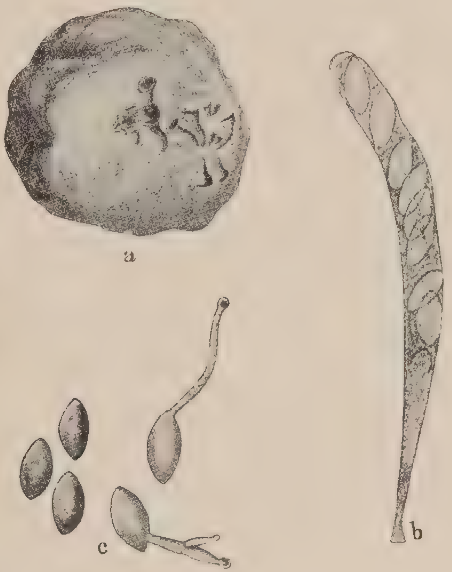

Fig. 318.—Brown Rot.
a, apple mummy with apothecia; *b*, ascus; *c*, ascospores. (After K. Aderhold and W. Ruhland.)

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Sclerotinia Aucupariæ Wor. develops on the leaves and fruits of the mountain ash (*Sorbus Aucuparia*). The disease shows itself in the form of dark brown streaks along the leaf veins, and from these points the fungus spreads to the rest of the leaf. Generally only individual leaves of the plant are attacked. The whole leaf finally becomes brown and withered. Next an ashen-grey, powdery covering of conidial pustules appears along the main veins and at the same time an unpleasant smell, resembling that of the mountain ash flowers, is noticeable. From these pustules the infection passes to the stigma of the flowers, the germ-tube grows down the style, and the ovary is finally changed into a brown, shrunken, dried-up, sclerotial mummy (Woronin, 1895). The disease has been found in Finland, Germany, and Switzerland. **S. Padi** Wor. appears on the under side of the leaves of *Prunus Padus*, with the

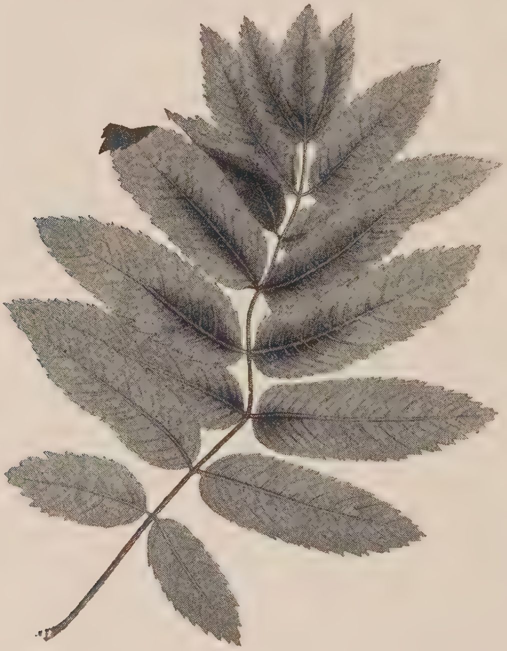

Fig. 319.—*Sclerotinia Aucupariæ.* (After M. Woronin.)

mycelium spreading along the main veins. The parts of the leaf surface that are attacked turn brown and die. The conidial pustules form a greyish-white covering on the leaves and stems. The conidia enter the stigmas of the flowers and the infected ovaries are changed into mummies. Apothecia grow out from the mummies in the following spring and the spores give rise to a fresh outbreak of disease (Woronin, 1895). **S. Cydoniæ** Schell. appears on *Cydonia vulgaris* (Schellenberg, 1899; Wormald, 1926); **S. Mespili** Schell. on *Mespilus germanica* (Schellenberg, 1906), and **S. Ariæ** Schell. on *Sorbus Aria* (Schellenberg, 1906; Wormald, 1926). These forms develop partly on the leaves of the host plants, generally on the under surface, and partly on and in the fruits, producing sclerotia and mummies (Schellenberg, 1923).

Sclerotinia Vaccinii Wor. attacks *Vaccinium Vitis Idœa.* The young whortleberry shoots shrivel in the spring, turn brown, and dry up. On the bark of the dead shoots there develops a matted, white layer of mould which by its pleasant almond-like scent attracts insects and in this way the conidia are carried to the stigmas of the flowers. The ovaries develop into white, inedible berries or finally into brown mummies which are hollow and open at the top and bottom. The mummies fall to the ground, over-winter there, and in the spring develop apothecia, the ascospores from which infect the young whortleberry shoots. The disease is widely spread in many European countries (Woronin, 1888). **S. baccarum** Rehm., a species very closely related to the foregoing. It develops a felt of conidia on the concave side of the bent stems of *Vaccinium Myrtillus* and causes the berries to be changed into white mummies (Ascherson and Magnus, 1891). **S. Rhododendri** Fisch. leads to the formation of sclerotia in the ovaries of *Rhododendron ferrugineum* and *Rh. hirsutum.*

Sclerotinia bulborum (Walck.) Rehm attacks the hyacinth crops (*Hyacinthus orientalis*), especially in Holland. Two forms of the disease are distinguished, a "White Rot" which commences at the neck of the bulb and spreads to the inside, and a "Black Rot" which causes premature yellowing and wilting of the leaves and often also prevents flowering. The mycelium produces black sclerotia which are found in large numbers as flat crusts on and between the scales. In the following year the sclerotia produce apothecia with a dark brown disk. If the infected bulbs remain in the ground they decay to a foul-smelling mass. Probably both forms are only different stages of the same species of fungus. The fungus also infects species of *Scilla* and *Crocus.*

Sclerotinia dioica Wor. & Naw. differs from the sclerotinia diseases previously mentioned in being heterœcious. The conidial stage is found on the shoots of *Vaccinium uliginosum.* The disease usually appears on a single leaf of the shoot, afterwards the whole shoot wilts and bends downwards. Ash-grey *Monilia* pustules break out on the diseased shoots, and the conidia are carried by wind or insects to the stigmata of *Ledum palustre* and give rise to sclerotia in the ovaries. After overwintering, apothecia grow out in the following spring, one apothecium from each sclerotium. The disease is found in Finland and Russia mainly; it has also been met with in several districts in Germany (Woronin, 1894).

Brown Rot of Stone Fruits

Sclerotinia cinerea (Bon.) Schröt.

Monilia cinerea Bon.

This fungus attacks cherries, plums, and peaches and the phases of development through which it passes are similar to those already described in the case of Brown Rot of apple and pear.

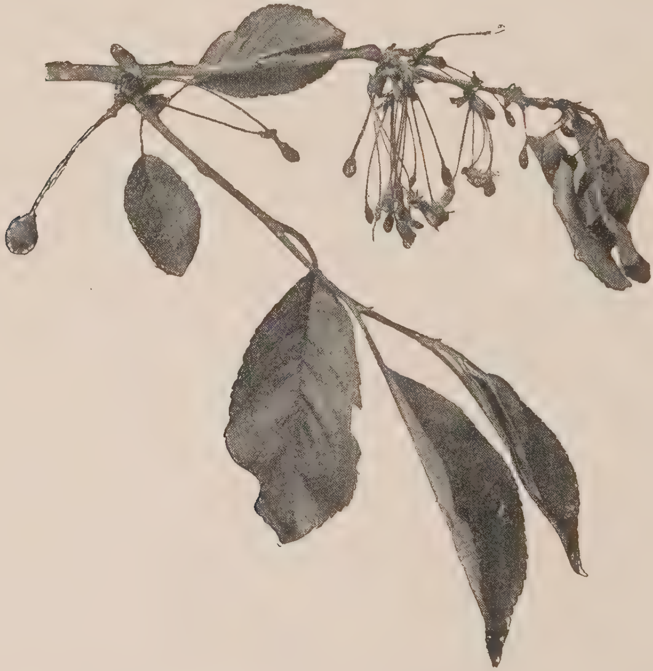

Fig. 320.—Brown Rot of Stone Fruits.

Twig of acid cherry, part of which has been killed by the Monilia stage of the disease. (Orig.)

On cherries, particularly acid cherries, the blossom and twig blight appears at the end of the flowering period. On a tree which has shown promise of a good crop, a number of flower trusses are seen to have a wilted, brown appearance and to be hanging down; near to them are groups of dead, drooping leaves. The tips of the shoots usually remain alive for a time and seem healthy, but gradually they die also. If the outbreak of disease is severe, most of the flowering trusses are affected and no fruit is produced. After several years of these severe attacks the tree itself may die.

The dead fruit trusses with the adjacent leaves do not, as a rule, fall immediately but remain, blackened and dry, throughout the summer in the head of the tree. If these dead trusses and leaves are left undisturbed they may remain on the tree over the winter and far into the next summer. A short time after the blossoms and twigs have died, it is often noticed that drops of gum, about the size of a pea or bean, are exuded in the angles of the dead branches.

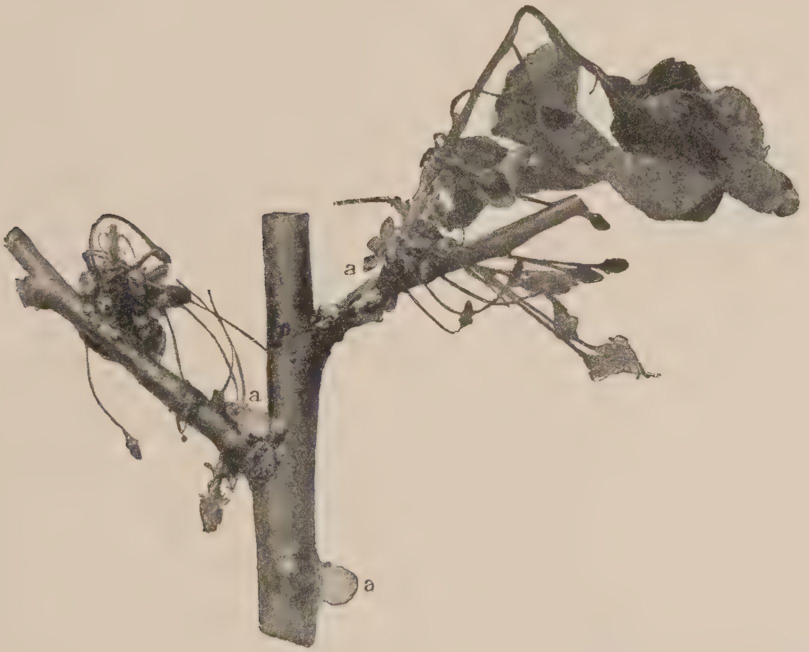

Fig. 321.—Brown Rot of Stone Fruits. Twig of acid cherry killed by the Monilia stage, exudation of gum (*a*) in the angles of the dead branches. (Orig.)

If at this period a careful examination is made of the dead flower and leaf tissues, above all on the outside parts of the flower stalks, it is found that here and there grey, wart-like pustules of hyphæ are present from which chains of conidia are abstricted (summer Monilia). On the thin flower stalks the pustules are extremely small, hardly visible to the naked eye, but they often coalesce and may have a length of 5–10 mm. immediately below the flower. On the remains of the flowers the pustules are generally a little larger and may attain the size of a pin's head.

The fungus over-winters in the stalks and twigs which remain on the tree and in the following spring gives rise to grey conidial pustules (previous season's Monilia) before the winter buds have opened. Conidia from these pustules infect the stigmata of the flowers which are shortly afterwards produced, and in this way another generation of grey conidial pustules (summer Monilia) arises. Thus the Monilia cycle, which covers the period of a year, is completed.

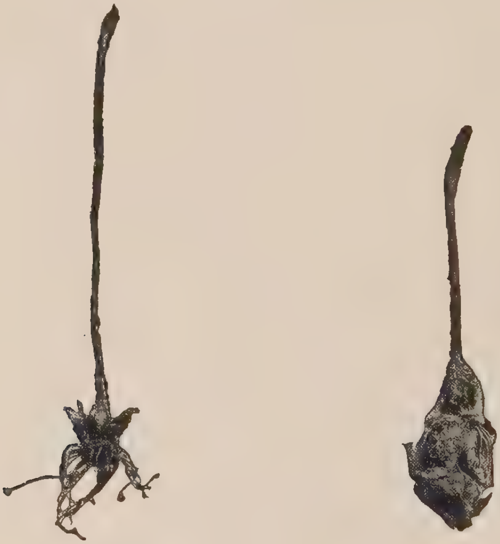

Fig. 322.—Brown Rot of Cherry. Conidial pustules on flowers. (Orig.)

There is also, as with Brown Rot of apple and pear, a "Sclerotinia cycle" with autumn Monilia followed by the formation of apothecia. The *Monilia* pustules show themselves on the fruit about the time of ripening, in the form of more or less closely-crowded, wart-like growths. The pustules require, as a rule, two over-wintering periods in the open before apothecia are developed from them in the spring. The spores formed in the asci of the apothecia give rise to an "autumn Monilia" when they are carried to young fruit structures.

The question arises as to how far the forms of fungus on the blossoms and twigs are identical with those on the fruit in the case of stone fruits (*Sclerotinia cinerea*) as well as in the case of those on the apple and pear (see *Fructigena*).

Firstly, the forms of disease on the fruits have been known for a much longer time than those on the blossoms and twigs. The former were known in the early part of the last century, whereas the latter were not observed until towards the end of the century. The earliest recorded cases of blossom and twig blight in Europe date from the years 1887 (Wurtemberg, Denmark), 1891 (Holstein, Prussia), 1894 (Berlin), and 1897 (in fifty-six different places in Germany). Secondly, it is certain that the blossom and twig blight overwinters without the assistance of the autumn stage on the fruit because, as described above, the previous season's stage of the fungus appears before the opening of the annual buds in spring on parts of the fruit and blossom which had died in the previous year. Thirdly, observations made in Sweden in 1905–07 point to the two forms of disease being independent of one another.

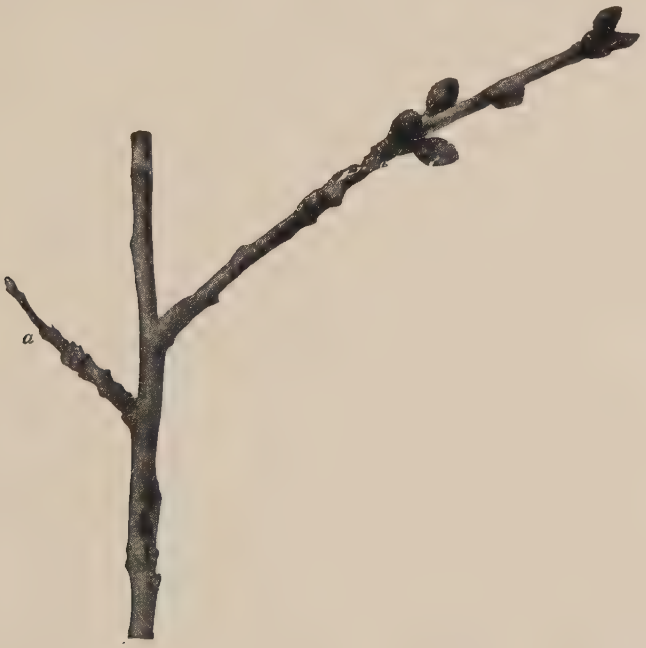

Fig. 323.—Brown Rot of Cherry. Twig attacked in previous year by Monilia stage; grey conidial pustules on the dead parts in spring (April). (Orig.)

In certain gardens where blossom and twig blight occurred severely, no diseased fruit was observed year after year.

In order to distinguish between the two forms of disease some inoculation experiments were carried out in Sweden in 1912. An inoculum of conidia from fresh *Monilia* pustules on the dead leaf-stalks of cherry was introduced into young cherries on the same tree; of twenty inoculations thirteen were positive. After 6–7 days fresh light-grey *Monilia* pustules appeared on the surface of the inoculated cherries. From the results of this experiment it is seen that the non-appearance of the autumn generation of the fungus on the cherries is not due to the inability of the summer generation to cause infection of the fruit. It is, however, surprising that such infection does not occur naturally in the open.

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Similar attacks of blossom and leaf blight are not infrequently found in early summer on *Prunus triloba*, *P. persica*, and *P. Padus*, the outbreaks on *P. triloba* often being very severe. Each year the number of flower trusses that is killed shows an increase. An interesting case in this connection came under observation during the summers of 1916 to 1921 in a garden in the south of Sweden. From two trees standing some 16 yards apart all the dead trusses and leaves were removed and destroyed, but each year the disease returned although less severely. In the summer of 1922 the whole of the head of one of the trees was cut off; in the following summer a new head grew out which, except for a few dwarf shoots in the lower part, was fresh and green. In early summer 1924 this tree carried splendid, healthy flowering branches.

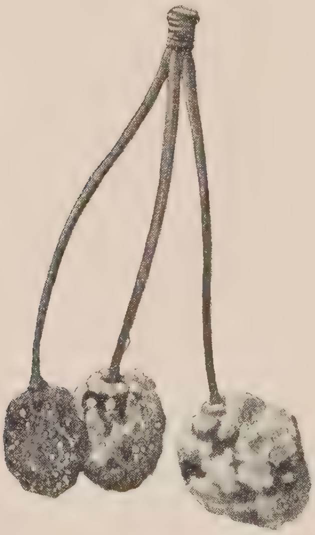

Fig. 324.—Brown Rot. Mummied cherries. (After O. Kirchner and H. Boltshauser.)

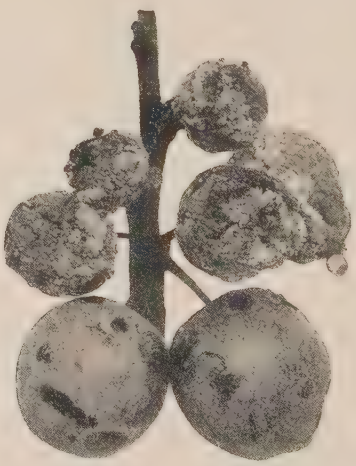

Fig. 325.—Brown Rot of Stone Fruits. Plums killed by disease. (Orig.)

A form of fungus which belongs here causes the death of the flowering twigs of *Forsythia intermedia* and *F. suspensa* about the end of the flowering period. Many of the petals are not thrown off after flowering but stick together and remain attached to the calyx ; in moist situations a growth of *Botrytis* occurs on them. The dying-back extends to the flower stalks, which in a short time become flaccid and hang over. Somewhat later, black sclerotia, about the size of a pea, are found in the diseased organs. After over-wintering in the open the sclerotia send out funnel-shaped apothecia. This disease appeared in the years 1902–05 in the garden of the Swiss Experiment Station at Wädensweil (Osterwalder, 1905).

A **Sclerotinia Disease of the Hazel Nut** (*Corylus Avellana*) is generally regarded as being due to the same species of fungus. The unripe nuts are noticed to fall at the end of June or beginning of July. The nuts are not separated at their pedicels but the whole fructification comes away when touched. Small, discoloured spots occur on the bracts (cupules) and on the surface of the nut itself there is a brown, annular, spreading discoloration. The disease begins, generally, at the tip of the nut, the cupules of which do not close completely but leave a narrow channel which leads to the interior of the fruit. Infection appears to take place through the stigma. In severe cases of the disease, the whole of the cupule, except a small basal portion, turns brown within a short time. The tissue of the kernel is of a deep brown colour and adheres firmly to the shell ; by the end of July the nuts are entirely brown and much shrivelled. *Monilia* pustules finally break out on the brown portions of the fruit wall and carpels. The fungus has been transmitted by artificial inoculation from the hazel nut to apples, plums, hips, and grapes.

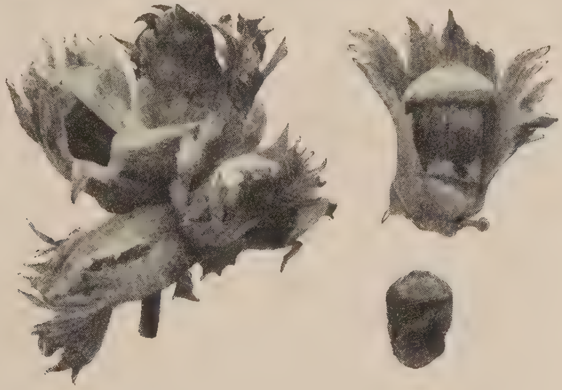

Fig. 326.—Sclerotinia Disease of Hazel Nut. Diseased nuts. (Orig.)

The fungus first appeared in Proskau in 1887 on several sorts of hazel nuts (Sorauer, 1900). In 1911–12 it caused considerable damage in Sweden, and in 1922 it was found in Austria (Wahl, 1924).

For the control of the disease, the same measures may be taken as in the case of Brown Rot of apples.

Vine Rot, Sclerotinia of the Vine.—In autumn, greyish-white *Botrytis* tufts appear on the leaves, shoots, and ripening grapes. The leaves are at first rust-coloured but gradually lose all colour. At the point of origin of the young shoots a brown swelling arises and a little later the shoot drops off. In dry weather the fungus causes the grapes to dry up and become like raisins ; the wine made from these dried grapes is often better than that from normal grapes. In damp weather the fungus generally brings about a wet rot of the grapes, and it may spread to the shoots and cause the crop to be considerably reduced. On the leaves, as well as on the grapes, sclerotia arise and they develop apothecia in the following summer. The sclerotia can be carried on the cuttings

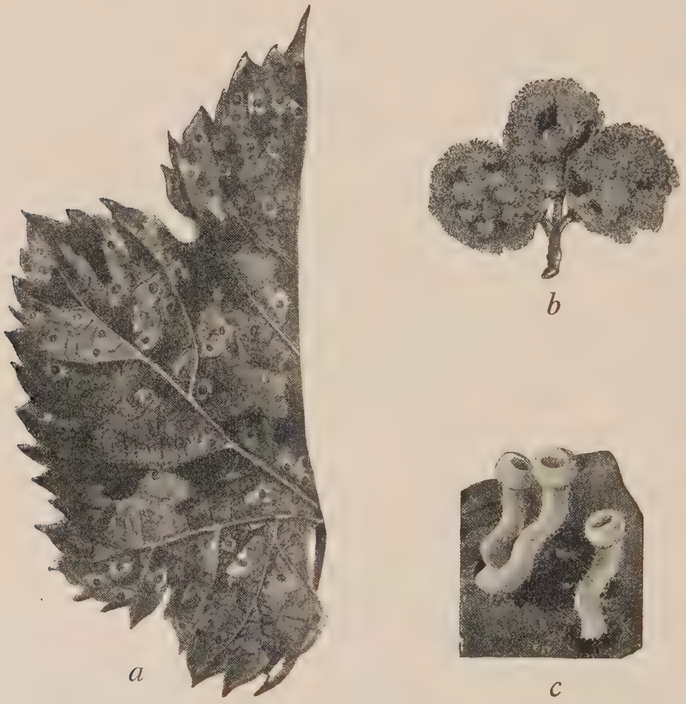

Fig. 327.—Vine Rot.

a, portion of leaf with sclerotia on the upper surface ; *b*, grapes covered with sclerotia ; *c*, apothecia. (After G. de Istvánffi.)

to the propagating beds, and if the latter have become infected they must be thoroughly disinfected (Viala, 1893 ; de Istvánffi, 1905).

Sclerotinia laxa (Ehr.) Aderh. & Ruhl.

Monilia laxa Ehr.

This fungus, formerly identified with *Sclerotinia cinerea*, attacks the apricot tree (*Prunus armeniaca*). Immediately after the tree has flowered, especially if the previous autumn was wet, large numbers of the flower- and leaf-trusses are seen to be hanging down, dead and black, on the tree—a true blossom and twig blight. Soon afterwards minute, grey, conidial pustules (spring Monilia) are discovered on the dead flowers and leaves, whilst at the same time large drops of gum appear on the neighbouring twigs and branches. After some weeks, the fruits which have developed from the flowers that were not infected are found to be covered with grey pustules (summer Monilia). Gradually these fruits are changed to greyish-black mummies, most of which remain attached to the tree during the winter, a few only falling to the ground. At the beginning of the following year, a winter Monilia develops upon the infected branches. It is in the form of small, grey pustules ; the conidia are viable until the end of April and so are able to infect the flowers as they are produced.

The mummied fruits finally fall to the ground, and after a resting period of 2–3 years apothecia are produced on the surface. In the stage of the disease on the fruit, this fungus has been known to science since 1818, and in the stage on the flowers and leaves since the eighties of the last century ; it was not, however, until 1903–04 that this form attracted much attention. The disease is found at present in Switzerland, France, Italy, Austria, and America, etc.

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Although the fungi which are the cause of the following diseases do not produce apothecia they can conveniently be dealt with here.

Botrytis Disease

Botrytis cinerea Pers.

Sometimes in summer, whitish or yellowish discolorations are observed at the base of the stem of rape plants (*Brassica Napus oleifera*). The cortex of the diseased patches lies loosely on the woody tissue and the epidermis can easily be peeled off. From the mycelium there is developed a dense, grey to brownish coating of simple or branched conidiophores at the tips of which ellipsoid conidia are abstricted in grape-like bunches. By means of the conidia the fungus spreads more or less rapidly to surrounding plants, according to the weather conditions. The fungus lives from year to year by means of hard, black sclerotia produced in the cavities of the stem; in spring these sclerotia send out fresh hyphæ and conidiophores.

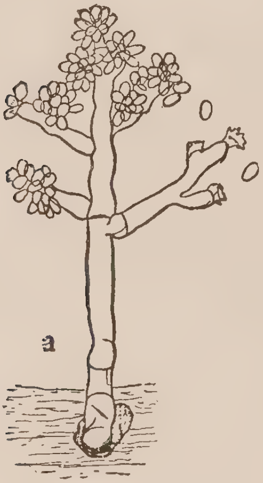

Fig. 328.—*Botrytis cinerea.* Branch of mycelium producing conidia. (After A. B. Frank.)

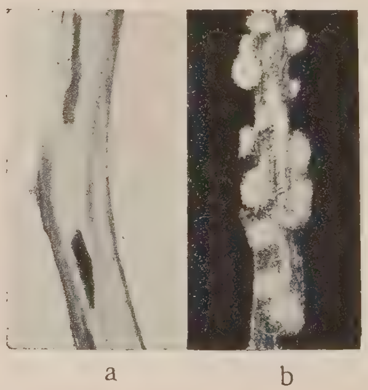

Fig. 329.—Botrytis Disease on Stem of Potato. *a*, stem with sclerotia; *b*, stem with *Botrytis* mould. (After G. H. Pethybridge.)

A similar disease attacks the stem of the hemp plant (*Cannabis sativa*). The hyphæ spread through the tissues of the stem into the medulla. In the intercellular spaces sclerotia are formed which after hibernation send out fresh *Botrytis* tufts in the following year. In the sclerotinia stage this form is also named *Sclerotinia Kaufmanniana* Trichom.

The *Botrytis* fungus, which often lives in association with the fungus *Sclerotinia sclerotorium* and receives from it considerable help in its work of destruction, is found on a number of cultivated and wild plants when external conditions—moisture, warmth, insufficient air supply, and low vitality of the plant—are favourable. The fungus ramifies in the young organs of the plant which it attacks, and obtains therefrom the nourishment which it requires to produce a branched, tree-like, aerial mycelium on which conidia arise in clusters. Sclerotia develop from the superficial *Botrytis* tufts and after hibernation they germinate with hyphæ.

Sometimes small, elongated, black, slightly raised, crust-like streaks appear on young potato stems in summer. These crusts, or scales, are composed of a dense network of hyphæ which develop into sclerotia from which small tufts of *Botrytis* arise.

As the Botrytis-form often appears in association with the Sclerotinia-form it was thought for a long time that *Botrytis* was only a conidial generation of apothecia-producing sclerotinia-forms. Recent investigations have shown, however, that this is not the case; *Botrytis* is an independent fungus the sclerotia of which after over-wintering develop *Botrytis* tufts but not apothecia.

Control Measures :

The same steps should be taken as in the case of Sclerotinia Rot.

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Botrytis Disease on Species of Ribes.—Often in summer the leaves of *Ribes Grossularia*, *R. rubrum*, and *R. nigrum* are seen to have acquired a brown to black colour on the tips and margins. This discoloration gradually spreads to the inner portion of the leaf lamina. Leaves attacked in this way usually fall prematurely and the bushes then become appreciably weakened. The leaf infection seems to take place through the water pores on the margins of the leaves. It is from the brown margins of the leaves that grey, conidial tufts are produced in damp situations. The fungus also appears on the berries, but not until the fruit approaches ripeness. Sclerotia develop on the diseased berries as well as on some tips of shoots which have not ripened their wood by the autumn. After over-wintering in the open, the sclerotia germinate and produce tufts of *Botrytis*.

Sometimes this fungus appears very destructively on *Ribes aureum ;*

thus in the years 1907–08 about 6,000 bushes of this variety were killed near to Stockholm by the *Botrytis* attack (Wulff, 1908).

Sclerotium cepivorum Berk. attacks a number of species of *Allium* such as *Allium Cepa*, *A. fistulosum*, *A. Porrum*, *A. sativum*, and *A. ascalonicum.* About the middle of July, if not earlier, small, whitish-grey *Botrytis* tufts appear on the lower parts of the young shoots as well as on the newly-forming bulbs. The leaves assume a pale, bleached appearance. On the bulbs the fungus appears as a white coating and somewhat later in the form of densely-packed, small, black sclerotia. Badly infected crops may be entirely destroyed, and the disease reappears each year on infected soil (Walker, 1924).

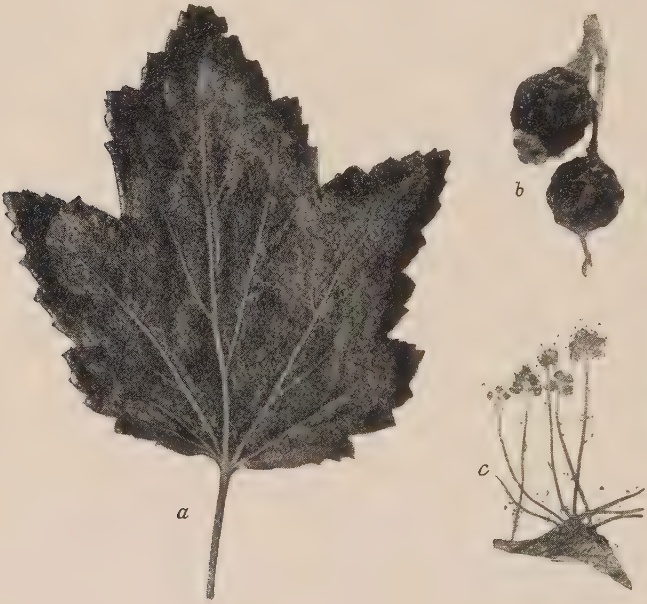

Fig. 330.—Botrytis Disease.

a, leaf of *Ribes rubrum*, margins of leaf turned brown by the disease; *b*, berries of *Ribes aureum* with tufts of *Botrytis*; *c*, conidiophores arising from leaf of gooseberry. (After T. Wulff.)

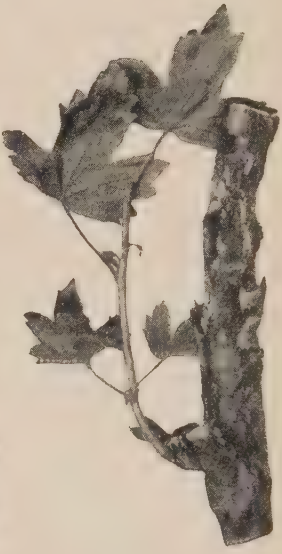

Fig. 331.—Stem of *Ribes aureum* with Botrytis Disease. (After T. Wulff.)

Botrytis Galanthi Ludw. produces a grey *Botrytis* growth on the leaves and flower parts of *Galanthus nivalis* as they appear above ground in spring, also black sclerotia on the bulbs; the diseased plants die completely after 2–3 years. This disease was very prevalent in the years 1915–17 in the south of Sweden (Malmö).

Botrytis Pæoniæ Ouch. produces black to ashen-grey spots on the bud scales, stems, leaf-stalks, and leaves of species of *Pæonia* and

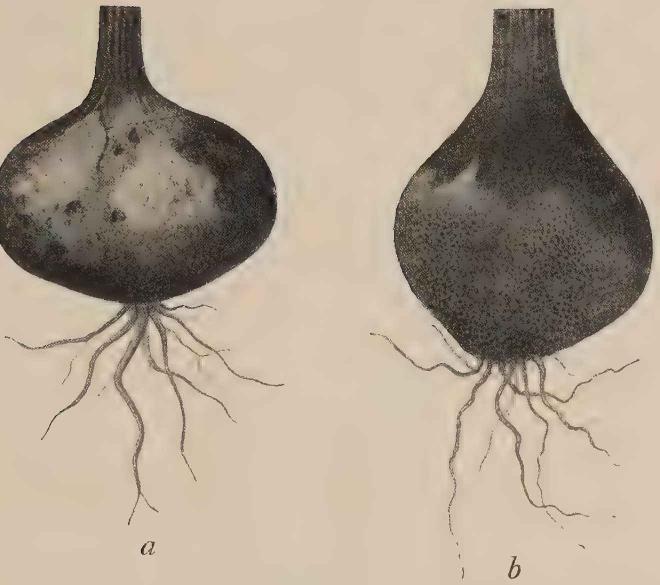

FIG. 332.—*Sclerotium cepivorum* on *Allium Cepa*.
a, early white stage ; *b*, later stage with black sclerotia. (Orig.)

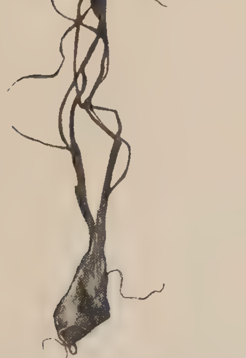

FIG. 333.—*Sclerotium cepivorum* on *Allium Cepa præcox*. (Orig.)

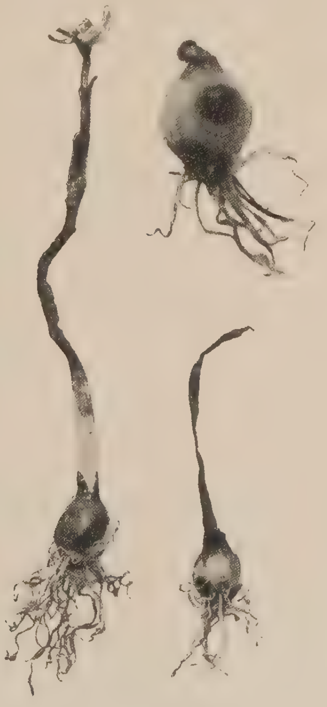

FIG. 334.—*Botrytis Galanthi*. (Orig.)

Convallaria. Thick coatings of conidia-bearing tufts of *Botrytis* develop on the diseased spots.

Botrytis Douglasii v. Tub. attacks the young developing shoots of species of *Picea* and *Abies* in summer and autumn. The needles grow abnormally and bend over, either in one direction or on all sides; the young stem in the meantime ceases to increase in length. The green colour of the needles changes to red, then brown, and finally yellowish-brown. On the surface of the browned needles, greyish-white hyphæ arise which sometimes form themselves to brush-like tufts on which colonies of *Botrytis* or small, black, shining, round or oval sclerotia are produced. From the sclerotia, conidiophores grow out in moist situations (v. Tubeuf, 1888; Behrens, 1895).

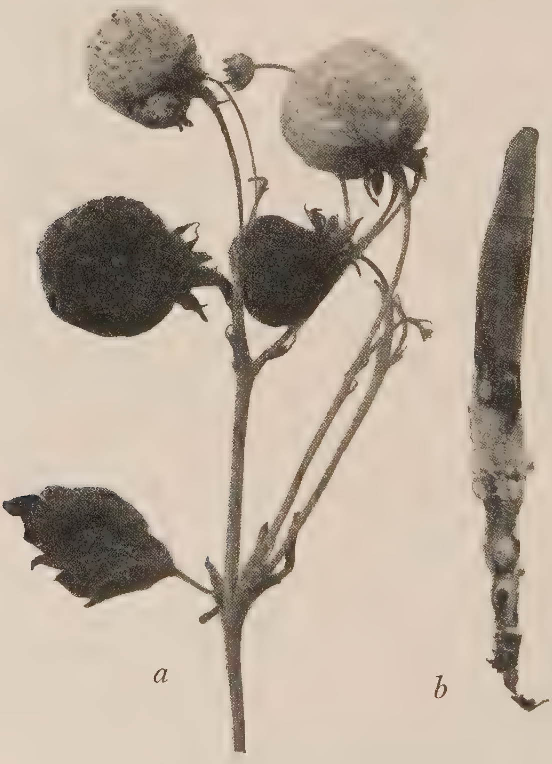

Fig. 335.—*Botrytis.*
a, on *Fragaria vesca*; *b*, on young bean pod. (Orig.)

Botrytis Disease on Cactus.—This was observed in 1918 on *Mamillaria centricirrha* and *M. elegans* in 1918 in a greenhouse in Berne. The surface of the stem became discoloured and sunken, the interior turning soft and slimy. On the surface of sections of the stem, white felts of

Botrytis appeared after the material had been kept for a few days in a moist place and some days later black sclerotia developed (E. Fischer, 1920). **Botrytis Diseases** are not uncommon on all kinds of vegetables and ornamental plants both out of doors and in the greenhouse. The parts that are attacked are seen to be covered with a grey, or white, mould arising from tufts of *Botrytis*. Eruptions of this nature are found on roses (on the undeveloped buds), strawberries (on the ripening fruits), beans (on young pods), on *Calendula*, etc. (Beauverie, 1899; Stevens, 1916).

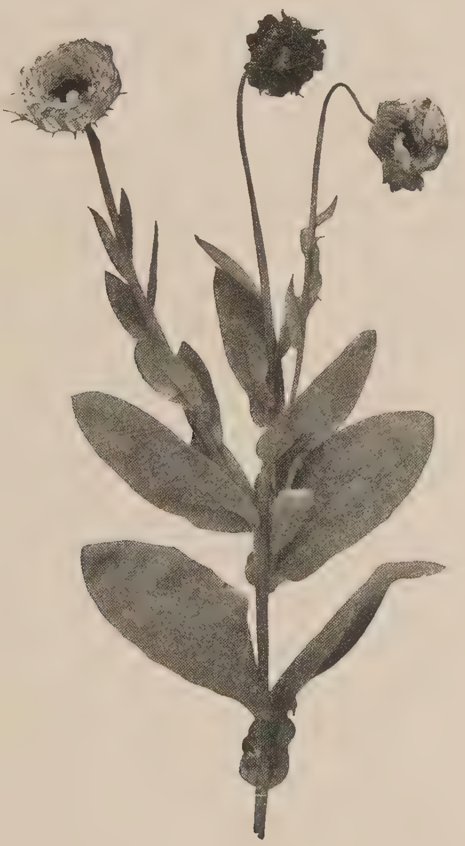

FIG. 336.—*Botrytis* on *Calendula officinalis*. (Orig.)

In North America (Alaska) similar diseases have been found on a large number of species of Phanerogams belonging to not less than 88 different genera (J. P. Anderson, 1924).

Botrytis Tulipæ (Lib.) Hopk. appears on *Tulipa Gesneriana*, *T. suaveolens*, and *T. sylvestris*. The bulbs either fail to grow or the shoot is killed before it reaches the surface of the ground. When the bulb is cut open the leaves as well as the stem have a hyaline, grey appearance and the inside is almost entirely decayed. On the outer, fleshy leaves, especially towards the apex of the bulb, a white fungous growth is found in which sclerotia are embedded. The sclerotia are white at first ; later they are dark on the outside. Sclerotia are also found in the surrounding layer of soil.

The disease also appears on fully-developed tulip plants, at first on the leaves as small yellow spots with dark margins. These spots cause the leaves to have a variegated, wrinkled appearance. Under moist conditions, there is a more or less considerable development of *Botrytis* tufts from the sclerotia and the spots on the leaves.

This fungus was first described in 1830, but it was not until more than fifty years later that it attracted attention. In 1880 it was reported from Italy and since 1900 it has been more generally observed : thus, in England 1901, North America 1902, Holland and Germany 1903.

In normal circumstances the fungus is limited to species of the genus *Tulipa ;* practically all varieties in this genus are susceptible. By artificial inoculation it has been possible to transmit the fungus to the leaves and stems of *Narcissus*, *Crocus*, *Hyacinthus*, and *Allium* (Klebahn, 1904 ; Hopkins, 1921).

Control Measures :

(1) Only healthy bulbs should be planted. (2) The bulbs should be carefully examined before planting ; if sclerotia are found on the outer scales they should be removed or else the scales cut off and burnt. (3) All diseased plants in the beds should be removed and burnt. (4) The storeroom should be cool and dry. (5) If possible a guarantee should be obtained from the grower that the bulbs are free from sclerotia.

Black Spot of Roses

Diplocarpon Rosæ (Lib.) Wolf.

Actinonema Rosæ (Lib.) Fr.

This disease gives rise to brownish-black spots, some of which are small, others large and usually of a circular form, on the upper surface of the leaves of the rose. On young leaves, radiating strands of hyphæ which are dichotomously branched towards the periphery, can be distinguished in the spots. The light brown colour of these hyphæ makes it easy to distinguish them against the brownish-black surface of the leaf. On older spots the dichotomous branching of the mycelial strands is only visible in the peripheral portions of the spots. The centres of the spots are covered with small, black pustules (*Asteroma radiosum* Fr. or *Actinonema Rosæ* Fr.). Numbers of the small leaves are rolled backwards and adhere so loosely that when the tree is shaken they fall to the ground. The whole bush may, within a short time, become defoliated except at the tips of the shoots where a few healthy leaves still persist.

In the young fungus-spots on the leaves the mycelium is found partly in the epidermis of the upper surface, immediately below the cuticle, and partly in the inner layers of cells. The mycelium disorganizes the tissues which it penetrates and causes the cuticle to rise in wart-like excrescences. At the apex of such a wart a hole finally appears through which the spores are extruded. The spores are two-celled and bent; they germinate easily in water, and only one of the two cells puts out a germ-tube.

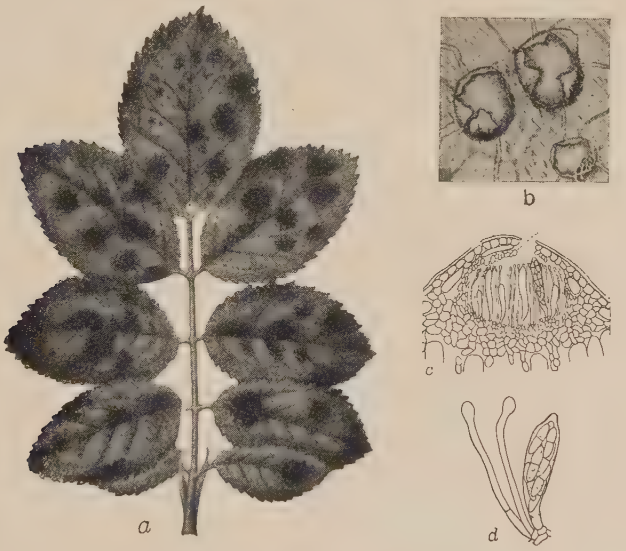

Fig. 337.—*Diplocarpon Rosæ.*

a, diseased leaf of *Rosa lutea* ; *b*, perithecia ; *c*, perithecium in section ; *d*, ascus and paraphyses. (*a*, Orig. ; *b*, after F. A. Wolff.)

After the infected leaves have over-wintered in the open, ripe perithecia appear towards the end of April or the beginning of May ; they are produced from the internal mycelium of the leaf. The ascospores are hyaline and two-celled, somewhat similar to the conidiospores ; they germinate well in a decoction of rose leaves.

Different varieties of roses vary in their susceptibility to the fungus even when growing close to one another ; very few sorts are immune.

The fungus was first reported and described from Sweden (E. Fries, 1815, 1828), later in Germany (1869), England (1877), and Hungary (1884). About the same time it was also found in the neighbourhood of Stockholm (Eriksson, 1885; Wolf, 1912; Klebahn, 1918).

Control Measures:

(1) Remove and destroy all diseased leaves in the autumn. (2) Spray the bushes with Bordeaux mixture in early spring before the disease appears on the leaves.

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Leaf Cast of Pine

Lophodermium pinastri (Schrad.) Chev.

This fungus attacks a number of species of pines such as *Pinus silvestris*, *P. austriaca*, *P. montana*, etc. In summer and autumn the needles of young, one- to four-year-old plants are marked by short, narrow streaks. These streaks are of a leaden-grey to yellowish-brown colour and are found chiefly on the under surface of the needles; they gradually extend and cause the needles to become red and dry. Older trees—seven to ten years—are more rarely attacked by the disease.

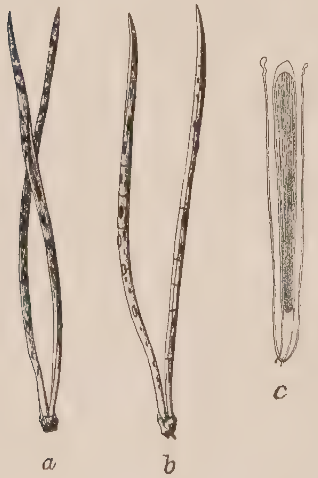

Fig. 338.—Leaf Cast of Pine. *a*, early; *b*, older perithecial stage; *c*, ascus and paraphyses. (*a* and *b*, Orig.; *c*, after P. Sorauer.)

In the diseased patches, pycnidia (*Leptostroma pinastri* Desm.) are produced in autumn with subepidermal stromata, from which narrow sterigmata arise and abstrict bacteria-like conidia. The epidermis is raised and afterwards ruptures, allowing the conidia to pass out. In winter, somewhat larger, black, shining apothecia develop on the dead needles which have fallen to the ground. These apothecia open by longitudinal fissures which close in dry weather and open in wet; the opening and closing of the fissure allows of the spores being freed gradually. The asci, as well as the paraphyses, are somewhat expanded at the tips; each ascus contains eight spores. Water is necessary for the formation of apothecia; when the pine needles are moistened they soon produce a luxuriant crop of apothecia. The ascospores are to be regarded as the means by which the disease is spread, infection taking place from the middle of July to the end of September. The pycnoconidia seem to be unable to transmit the fungus.

The various species and varieties of pine differ in the degree to which they suffer from this disease, as do also the varieties grown in different countries. The most susceptible is *Pinus austriaca*, whilst *P. montana* and *P. Strobus* are more resistant.

Control Measures :

(1) Only the best and most vigorous seedlings should be grown on. (2) Nurseries should be situated as far away as possible from places where the disease occurs and suitably sheltered sites should be selected. (3) As soon as the first apothecia begin to open, usually from the middle of July to the middle of August, the trees should be sprayed with a suitable copper fungicide.

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Haak. Der Schüttepilz der Kiefer. Zeitschr. Forst- u. Jagdw., 1911, 329–357, 402–423, 481–505.

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Lophodermium macrosporum (R. Hart.) Rehm. occurs on the needles of *Picea excelsa.* The needles of the previous year's shoots turn brown in the spring, often all the needles on a shoot, and on them apothecia are produced in the course of the summer. The apothecia are long, straight, shining, black swellings, one to three being found on each half of the under side of the needle. The diseased needles remain, as a rule, on the withered shoots, and in such cases the apothecia only ripen in the following spring and then eject their ascospores. It sometimes happens that the needles suffering from the disease in spring fall to the ground in the first summer and there develop and ripen their apothecia. In other cases the browning of the two-year-old shoots first takes place in the autumn, and another winter is required for the ripening of the apothecia; the spores are then freed in summer. Subepidermal masses of conidiophores occur as well as apothecia. This disease seems to prefer to attack trees that are from 8–20 years old (v. Tubeuf, 1895). **L. nervisequium** occurs on *Abies pectinata.* Pycnidia appear in early summer on the two-year-old needles in the form of black, undulating, longitudinal swellings. In the next year, apothecia are found in swellings running along the midrib ;

the apothecia ripen in April of the following year. In each ascus are eight spores, four in the upper and four in the lower part (v. Tubeuf, 1895). **L. Abietis** Rostr. on *Abies excelsa* and *A. pectinata*. The disease appears on the needles of the two-year-old shoots of ten- to twenty-year-old trees, at first as pale, transverse bands and single, dark spots, then as dot-like, black pycnidia and large, black, elliptical apothecia and narrow, dark, transverse streaks. The diseased needles usually fall prematurely. The disease is widely spread in Denmark (Rostrup, 1902). **L. brachysporum** Rostr. on old, as well as on young trees of *Pinus Strobus*. In summer the needles turn brown and fall, the apothecia are short and black, the asci and spores short and broad, the latter surrounded by mucilage. The disease has been very destructive in Denmark (Graves, 1913). **L. juniperinum** Fr. on *Juniperus communis*, *J. virginiana*, and *J. Sabina ;* scattered needles are attacked ; sometimes individual twigs or whole branches are killed. **L. Laricis** Tub. occurs on the larch and is characterized by numerous yellow tufts of needles on the lower twigs. The needles die and fall ; a row of very small, black apothecia occurs in the middle of the needle (v. Tubeuf, 1895).

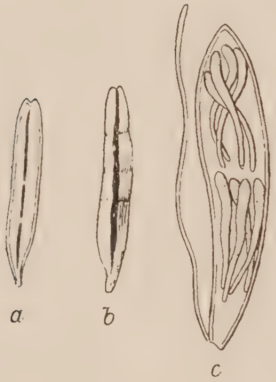

FIG. 339.—*Lophodermium macrosporum.*

a, spruce needle with pycnidia on the upper side ; *b*, needle with apothecia ; *c*, ascus and paraphysis. (After E. Rostrup.)

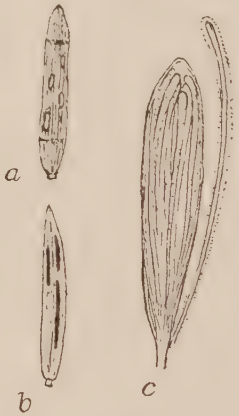

FIG. 340.—*Lophodermium nervisequium.*

a, fir needle with narrow swelling on upper side ; *b*, needle with broad swelling on under side ; *c*, ascus and paraphysis. (After E. Rostrup.)

Cenangium Abietis (Pers.) Rehm, also named *C. ferrugineum* Fr., attacks the stem-parts of *Pinus silvestris*, *P. austriaca*, *P. montana*, *P. laricio*, *P. rigida*, and *P. tuberculata*. The mycelium penetrates the bark and causes the death of the youngest shoots on trees aged five years and older. The infection of the shoots takes place below the terminal buds after the winter resting period is over and the cells are in their most active state. The needles on the affected twigs change to a red colour and fall prematurely; older branches are also attacked. The fructifications of the fungus are found on the older twigs, especially on the dead tissues, and in wet weather they appear as leathery membranes; in years favourable to their development very large numbers are produced. The conidia are of two kinds, one being unicellular, rod-shaped (*Dothichiza ferruginosa* Sacc.) and the other multicellular, sickle-shaped (*Rhabdospora pinea* Karst.). The apothecia break out in small groups on any part of the bark and are arranged in longitudinal or diagonal rows; they are of a leathery nature and when closed are of a black or dark brown colour. The hollows of the apothecia are furnished with paraphyses. The ascospores are ellipsoidal, one-celled and colourless, the paraphyses clavate (Frank Schwarz, 1895; v. Tubeuf, 1903; Lagerberg, 1913; Weir, 1921).

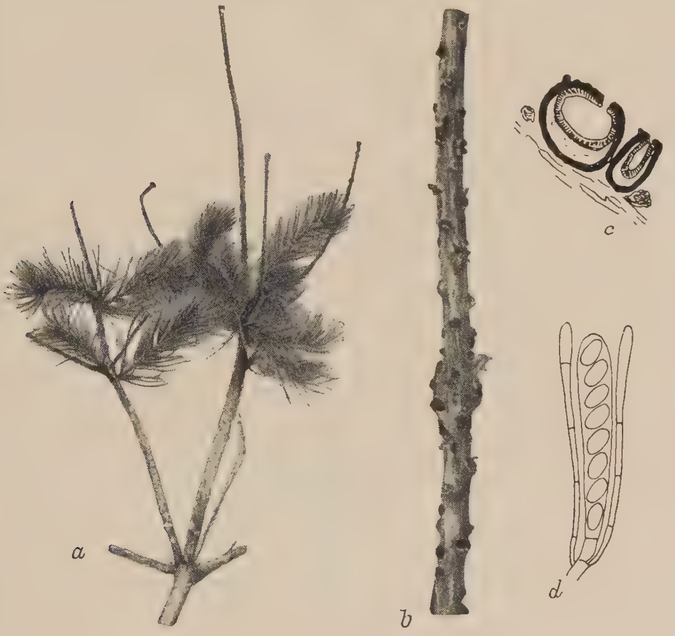

FIG. 341.—*Cenangium Abietis.*

a, dead tip of pine tree and dwarfed shoots; *b*, apothecia breaking through the bark; *c*, apothecia; *d*, ascus and paraphyses. (*a* and *b*, after Fr. Schwarz; *c* and *d*, after F. W. Neger.)

It has been suggested that **Brunchorstia destruens** Eriks., which has been observed in Scandinavia on certain species of pine such as *Pinus austriaca*, *P. montana*, etc., may be a pycnidial stage of the fungus described above. but at present this appears doubtful.

Dermatea carpinea (Pers.) Rehm. occurs on the stem and branches of *Carpinus Betulus*, the small branches often dying unilaterally. The mycelium lies below the bark and produces pustules which burst through the bark and liberate conidia. The conidia are of two kinds—ellipsoidal and rod-shaped. Later on, the stroma produces apothecia. The ascospores are one-celled at first, later, two- to four-celled. **Hysterographium Fraxini** (Pers.) de Not. occurs on the ash. Hard, elliptical, sunken patches occur on the surface of the stem. On these patches both pycnidia and apothecia are found; the asci have muriform, divided spores which are ultimately of a dark-brown colour. The fungus has appeared very destructively in Denmark (Rostrup, 1883).

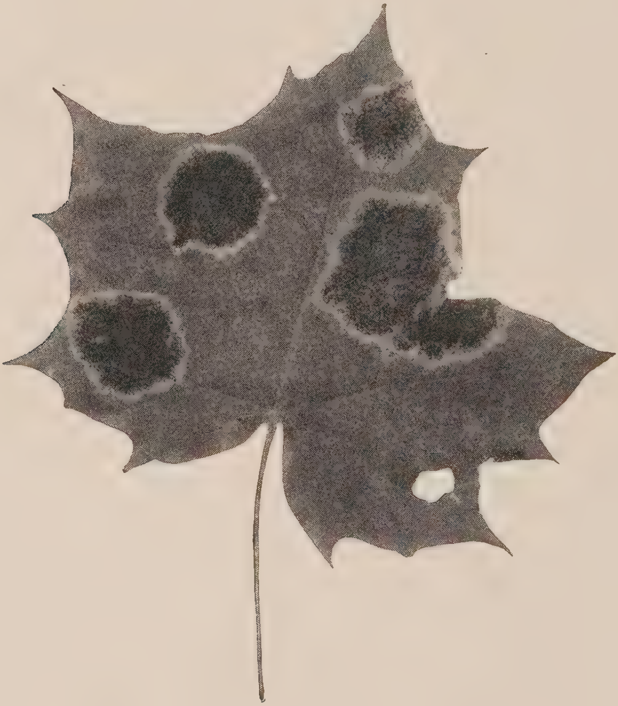

Fig. 342.—*Rhytisma acerinum.* (Orig.)

Rhytisma acerinum (Pers.) Fr. forms, from July or August onwards, large, round, black spots with light margins (sclerotia) on the upper surface of the leaves of species of *Acer*. In these spots lie subcuticular stromata from which conidiophores arise (*Melasmia acerina* Lév.); the diseased leaves usually fall prematurely. The spots on decayed leaves which have over-wintered on the ground are seen in spring to be swollen and wrinkled. In damp weather the pale-coloured apothecia appear, and their spores, which are surrounded by mucilage, are expelled when the trees come into leaf.

There are a number of specialized forms of this fungus: (1) f. sp. platanoidis K. Müll., the common form with large spots which attacks particularly *Acer platanoides* and *A. campestris* and rarely *A. Pseudoplatanus*; (2) f. sp. campestris K. Müll, attacks chiefly *Acer campestre*, often also *A. platanoides*, but never *A. Pseudoplatanus*; and (3) f. sp. Pseudoplatani K. Müll, a form with small spots which is strictly limited to *A. Pseudoplatanus*.

Infection takes place through the stomata on the under side of the leaf if the parenchyma of the upper surface has not been exposed through some mechanical injury.

The severity of the attack depends partly upon the conservation of the sclerotia, partly, and indeed mainly, upon sufficient moisture at the time the spores are liberated in April and May. The fungus is not a serious danger to forestry because even when the outbreaks are severe there is a satisfactory formation of wood (K. Müller, 1912; v. Tubeuf, 1913).

Rhytisma salicinum Pers. occurs on the upper surface of the leaves of a number of species of willow such as *Salix capræa*, *S. cinerea*, and *S. aurita* as thick, black, shining sclerotinia patches with yellow edges. **Cryptomyces maximus** Fr. produces broad, smooth, black crusts with yellow margins on the bark of the twigs of *Salix incana*, *S. purpurea*, *S. alba*, *S. lanceolata*, etc. The crusts swell when wetted by rain, and on drying they peel off, leaving long scars. The apothecia have cylindrical asci, one-celled spores, and thread-like paraphyses. The twigs die off above the diseased parts (v. Tubeuf, 1895). **Clithris quercina** Pers. occurs on the branches and twigs of the oak. The fructifications are straight or bent either diagonally or at right angles to the long axis of the twig. The ascospores are filiform, extruded in clots, and assume a circular form (Neger and Dawson, 1907).

Brusca Disease of the Olive Tree.—A diseased condition of the olive tree characterized by the wilting of the leaves has been known in several districts in Italy since the middle of the eighteenth century. The disease has been named *Brusca parasitica*, but recent investigations have led to the view that the fungus *Stictis Panizzei* de Not. is usually the causal organism, especially in the provinces of Lecci (Apulia) and Sassari (Sardinia).

The disease is first seen at the beginning of November on the older leaves, which change to a greyish-red colour in 8–10 days. The upper surface of the leaf, and afterwards both surfaces, is "marbled" with irregular, brick-red to copper-brown patches. On these patches, which finally turn white, blackish pycnidia soon develop on the under surface, and later, in the course of the winter (December to February), apothecia appear on the upper surface. The apothecia contain asci, each ascus with eight spores. The diseased leaves remain on the tree as a rule, only those which have been attacked near the end of the leaf-stalk being shed prematurely.

The mycelium lives during the summer in the tissues surrounding the dead leaf patches, and after the first autumn rain it produces a reddish ring of primary pycnidia, the spores from which give rise to secondary pycnidia. The olive leaves are infected in autumn by the pycnospores; neither the one-year shoots nor the young buds are infected. Warm, damp, misty weather appears to be favourable to the growth of the mycelium and to lead to an extensive formation of pycnidia. Trees of weak growth and with a badly developed root system are very susceptible to the disease.

The Brusca disease checks the growth of the branches and sometimes also hinders the formation of blossom. If, as a result of the disease, the foliage has completely withered for two or three consecutive years, the tree ceases to produce fruit.

None of the varieties of olive is quite immune, but certain sorts belonging to the group Cellina (Scurannese, Rosciola, Nardo, Casciola) are relatively resistant, whereas the variety Ogliarolo, long valued on account of its fruitfulness and the high oil-content of its fruit, is very susceptible and has had to be largely replaced by the Cellina varieties. These latter, when grafted on to Ogliarolo, have retained their resistance.

The disease was first observed at the beginning of the present century in Liguria and Northern Sardinia; it has since spread to other districts in Italy (Moschettini and Presta, 1777; Brizi, 1903; Cuboni, 1905; Petri, 1905; Peglion, 1912).

In Mid-Italy there is another form of Brusca disease which is due partly to the attack of *Cryptoascus oligosporus*, which injures the roots, and partly to the disturbance of the assimilation of the leaves by several fungi such as *Coniothyrium Oleæ* Poll., *Hormiscium Oleæ* Cast. and species of the genus *Phyllosticta*.

Control Measures:

(1) Spray the under surface of the leaves at the beginning of October with Bordeaux mixture. (2) Encourage root development by appropriate methods of cultivation.

Pseudopeziza Medicaginis (Lib.) Sacc. appears on *Medicago sativa*, *M. lupulina*, *M. falcata*, etc., and also on species of the genera *Melilotus*, *Onobrychis*, *Trigonella*, and *Vicia*. The fungus causes numerous round, dark spots on the upper surface of the leaf. In the centre of the spot is a brownish-black, waxy disk (apothecium) with an irregular, jagged margin. The disk is covered with closely crowded hyphæ intermingled with paraphyses. In severe outbreaks of the disease, all the leaves wither prematurely and fall. The fungus over-winters on dead leaves, the ascospores which arise therefrom cause infection in the following year. The fungus is found throughout Europe, sometimes causing much damage ; in America it is regarded as the most serious leaf disease of lucerne (Jones, 1919) ; a conidial stage has not been observed. **P. Trifolii** (Bernh.) Fuck. is closely related to the above lucerne-fungus ; in fact, until recently it was regarded as identical with it. The clover-fungus is also widely disseminated, but in general it appears to be less destructive than the lucerne-fungus.

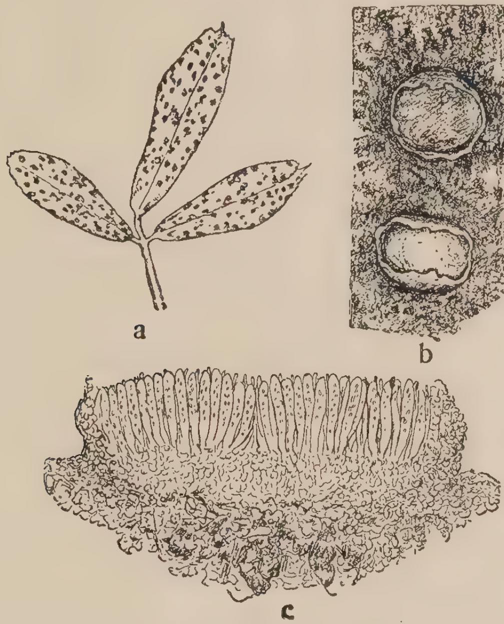

Fig. 343.—*Pseudopeziza Medicaginis.*

a, leaf with spots of fungus ; *b*, two fructifications with reflexed margins ; *c*, fructification in section, with numerous closely crowded asci. (After E. Prillieux.)

" Roter Brenner " of Vine

Pseudopeziza tracheiphila Müll.-Thurg.

This disease occurs on the leaves of the vine and gives rise to large discoloured patches in the angles of the veins. In the case of vines bearing purple grapes, the patches are red, bright green, or yellowish, whereas on white grapes the patches, which are at first of a yellowish colour, almost white, become bright red to brown when the leaf dies. A longitudinal section shows the mycelium of the fungus (*Pseudopeziza tracheiphila* Müller-Thurg.) in the vessels of the discoloured veins ; the hyphæ are sparsely septate, little branched, and curved in a characteristic serpentine fashion. The walls of the vessels, as well as those of the adjoining cells, are coloured brown to red and the chlorophyll granules are disintegrated.

Although the fungus is confined to the inside of the vessels of an infected, but still living, leaf it can, when the leaf dies, pass to neighbouring tissues and there live saprophytically and extend. If the old leaves lying on the ground in a vineyard where the disease was present in the previous year are examined in early spring it is found that most of them—up to 90 per cent.—bear apothecia. These apothecia develop fully in the spring, earlier or later according to the weather conditions, and the spores which are ejected infect the new leaves. In late autumn, segmented and branched hyphæ with conidiophores and spores are produced from the mycelium in the old disease-patches.

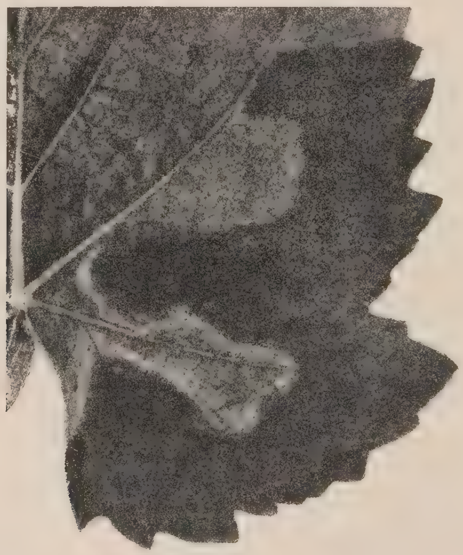

FIG. 344.—*Pseudopeziza tracheiphila*. Vine leaf attacked by the fungus. (After H. Müller-Thurgau.)

From old reports it is known that the disease has been present for a long time in European vineyards; probably it is now to be found in all vine-growing countries in Europe. In various districts in Switzerland, on the German side of Lake Constance and in France the vine Blue

Burgundy or Clevner (Pinot noir) has proved very susceptible. Other sorts which are severely attacked are Räuschling, Portugieser, and Erlenbacher on Lake Zurich; the white and purple Veltliner in Austria; the Riesling and Sylvaner in Germany; the Gamay, Hybrides Bouchet, and Teinturier in France. None of the European vines appears wholly immune.

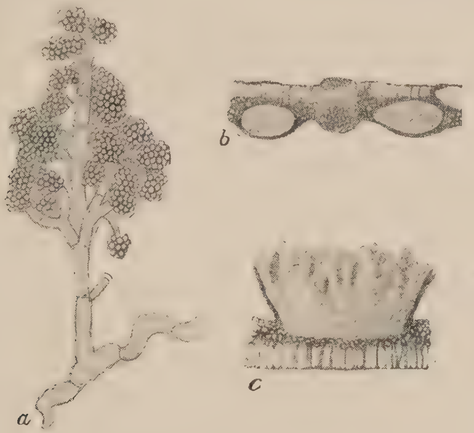

Fig. 345.—*Pseudopeziza tracheiphila.*

a, conidiophores; *b*, cross-section of over-wintered diseased leaf with closed apothecia; *c*, apothecium. (After H. Müller-Thurgau.)

Control Measures:

(1) Sandy or gravelly soils through which rain-water rapidly percolates should be heavily dunged. (2) On heavy loams and clays where the roots are not able to penetrate deeply, the soil should be improved by draining, by the application of such manures as open up the soil, and by appropriate cultivation. (3) Resistant varieties of vines should be planted. (4) Spray with Bordeaux mixture, the first application at the end of May or beginning of June and further applications as required.

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Müller-Thurgau, H. Der rote Brenner des Weinstockes. I. Zentr.-Bl. Bakt., Abt. 2, Bd. 10, 1903, 38 pp.; II. Bd. 38, 1913, 36 pp.

Pseudopeziza Ribis Kleb. occurs on the leaves of currant bushes in the form of discoloured patches from which small, pale fructifications arise. The spores are one-celled. The conidial stage is named *Glœosporium Ribis* (Lib.) Mont.

Pyrenopeziza Medicaginis Fuck. produces elongated, yellow patches along the veins of lucerne leaves; the mycelium ramifies in the inner tissues ot the leaf. After a time, open pustules are formed on the discoloured patches, and from the bottom of the pustules large numbers of conidia develop. This conidial stage has been known for a long time as *Phyllosticta Medicaginis* (Fuck.) Sacc. When the conditions are favourable, numbers of small, black bodies arise in autumn on the under surface of the leaf and they are changed into apothecia after the leaf dies. The disease is more or less destructive in a number of European countries (Germany, Austria, France, Italy, etc.), but above all in America (Jones, 1918).

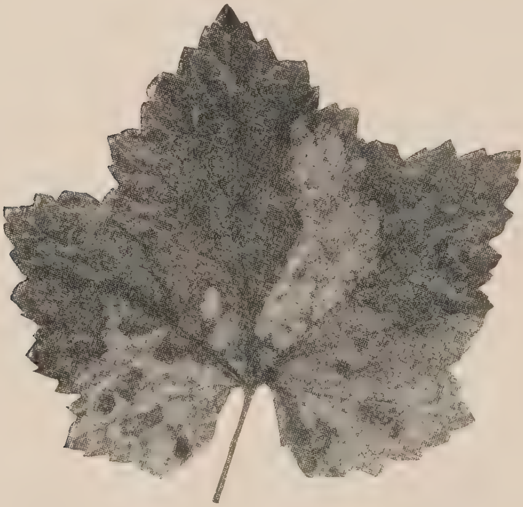

Fig. 346.—*Pseudopeziza Ribis.* Leaf of *Ribes rubrum* attacked by the fungus. (Orig.)

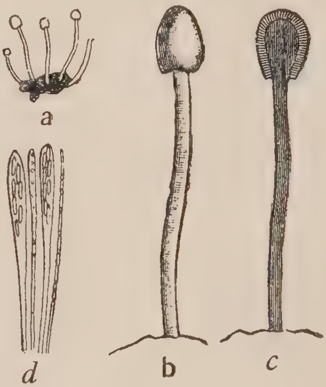

Fig. 347.—*Mitrula sclerotiorum* on Clover.

a, sclerotium with five fructifications; *b*, a fructification; *c*, fructification in section; *d*, asci from the surface layer of the fructification. (After E. Rostrup.)

In this connection, mention may be made of **Mitrula sclerotiorum** Rostr. which sometimes attacks species of clover, birdsfoot trefoil and other related *Leguminosæ.* The attack of the fungus is similar to that of clover rot (*Sclerotinia Trifoliorum*), and there is a production of sclerotia just as with the latter fungus. From the sclerotia there grow out, after a resting period, five to six, sometimes as many as thirty, white, later flesh-coloured. fructifications with a thin stalk and a club-shaped swollen head. This fungus is found particularly on species of *Lotus* in Denmark (Rostrup, 1902).

CHAPTER XVI

FUNGI IMPERFECTI

THE life-cycle of the forms of fungi which are placed in this group is not completely known.

Sphæropsidaceæ

The conidia of these fungi are enclosed in special globose pycnidia which are visible to the naked eye as minute brown or black dots. The pycnidia have usually a small, round, apical opening through which the conidia are extruded in the form of a long slimy tendril.

Phyllosticta

The pycnidia appear as crowded, brownish-black dots in the middle portion of the leaf-spots which are sharply defined and often surrounded by a dark red or brown ring. The conidia are ellipsoidal, one-celled, and colourless. The fungi belonging to this genus generally cause puncturing of the leaves and premature withering, with the result that the productive capacity of the plant is reduced.

A large number of species belong to this genus, and they are distinguished from one another, mainly, by their occurrence on various host plants.

Phyllosticta Tabaci Pass. causes light-coloured, dry spots on the leaves of the tobacco plant. The spots sometimes show dark punctations in the middle and are usually surrounded by a dark ring.

Other forms of *Phyllosticta* which may be mentioned are : **Ph. Trifolii** Rich. on species of clover, **Ph. Anthyllidis** Baud. on kidney vetch, **Ph. Fabæ** West. on horse beans, **Ph. phaseolina** Sacc. on other beans, **Ph. Polygonorum** Sacc. on buckwheat, **Ph. Humuli** Sacc. & Speg. on hops, **Ph. Cannabis** Speg. on hemp, **Ph. Mali** Prill. & Delacr. and **Ph. Briardii** Sacc. on apples, **Ph. piricola** Sacc. & Speg. and **Ph. pirina** Sacc. on pears, **Ph. prunicola** Sacc. and **Ph. cerasicola** Sacc. on cherries, **Ph. Persicæ** Sacc. on peaches, **Ph. Beijerinckii** Vuil. on stone fruits in general, **Ph. fragariicola** (Fr.) Sacc. on strawberries, **Ph. Rosæ** Desm. on roses and raspberries, **Ph. Cyclaminis** P. Brun. on *Cyclamen persicum*, **Ph. Dianthi**

West. on carnations, **Ph. Violæ** Desm. on violets, **Ph. salicicola** v. Thüm. on willows, **Ph. populina** on poplars.

Phoma

The pycnidia appear on the stems and roots and only exceptionally on leaves, especially conifer needles. The stem-parts when attacked are generally strictured, and in consequence the portion above the point of attack is killed owing to the failure of nourishment (twig blight). The conidia are usually one-celled.

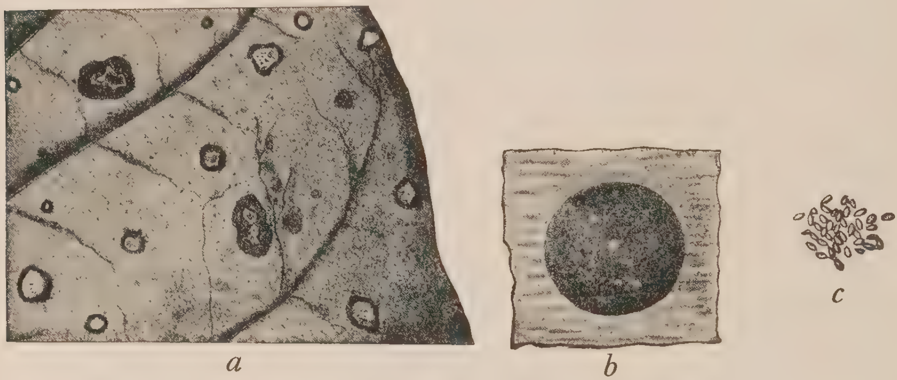

Fig. 348.—*Phyllosticta Tabaci.*
a, piece of tobacco leaf with fungus spots ; *b*, pycnidia in the middle of one of the spots ; *c*, conidia. (After O. Kirchner and H. Boltshauser.)

Black-Leg of Cabbage

Phoma oleracea Sacc.

This disease appears in somewhat varying forms at different times of the year.

One form shows itself on growing cabbage plants by the dying off of the main root immediately below the surface of the soil. All the soft parts of the roots are destroyed and only the firm vascular bundles remain. From the stem, immediately above the dead portion, numerous supplementary roots are put out, and if these develop strongly at an early stage the plant may live for a time, otherwise it dies. The disease can attack very young plants, those with only three or four leaves, and such plants are noticeable on account of their stiff, erect leaves. Red cabbage and cauliflower, sometimes other varieties, suffer most, whereas savoy cabbage only rarely. Plants attacked by this fungus sooner or later collapse.

Another form of the disease appears in winter on stored cabbages. Small spots, which at first are white, later greyish-brown and finally brownish-black, are found within the stem. These canker-like lesions extend, and the disease spreads through the leaf-stalks to the outer leaves and finally to the inside leaves. A warm, damp atmosphere favours the spread of the disease, and from January onwards it proceeds rapidly, the leaves dying from below and dropping from the stem. A downy mycelium is often found on the surface of the affected parts and usually also a large number of small dots which at first are red and later brownish-black. The latter are the pycnidia of the causal fungus, *Phoma oleracea*. The cell tissues of the diseased parts of the plant are permeated by the mycelium of the fungus.

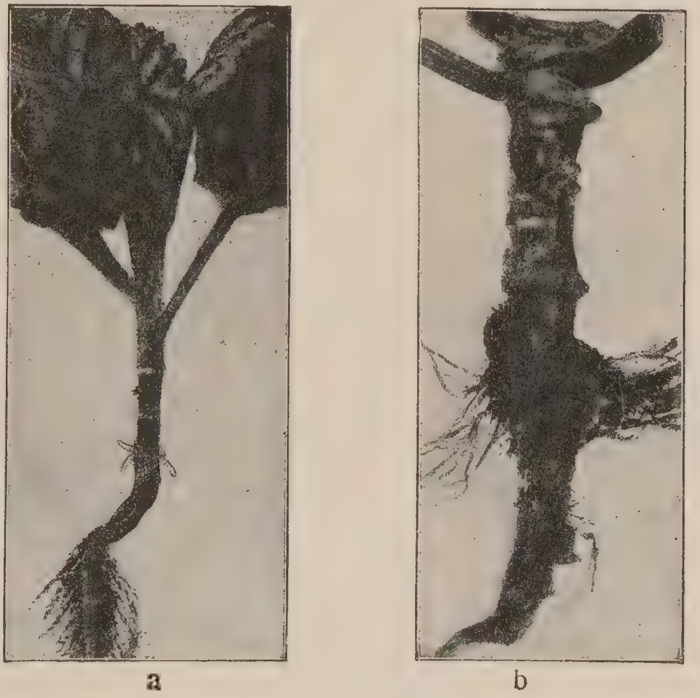

Fig. 349.—Black-Leg of Cabbage.

a, young cabbage plant in early stage of disease ; *b*, older plant from which leaves have fallen and fresh supplementary roots have developed. (After J. Ritzema-Bos.)

Investigations have shown that the two forms of disease described above are only different stages in the development of the same fungus.

In addition to the fungus there is, however, an insect, the cabbage fly (*Anthomyia Brassicæ*), at work ; indeed, when the disease occurs on the growing plant, it appears that the insect is the forerunner of the attack and enables the fungus to find entrance.

It has not been possible to prove that the disease is carried in the seed, but it has been observed that seeds from different sources have produced plants which have differed in their power of resistance to the disease. In certain districts in Holland where cabbage is grown on a very large scale, the disease has occasioned much damage for a number of years past; it has also been found in Germany and Italy.

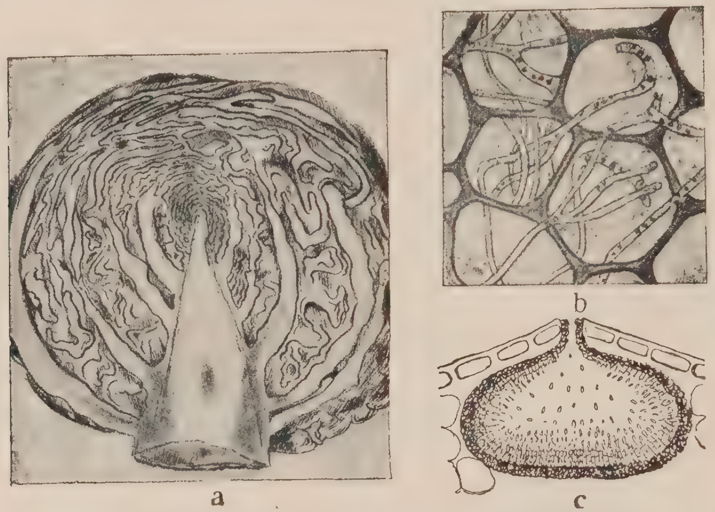

FIG. 350.—*Phoma oleracea* on stored Cabbage.

a, head of cabbage, with canker-like lesions; *b*, cells of diseased tissue with hyphæ; *c*, pycnidia with conidia. (After J. Ritzema-Bos.)

Control Measures:

(1) The young plants when drawn from the seed-bed should be washed with water and the adhering earth carefully removed from the stem and the base of the leaves. By this means any eggs of the cabbage fly which may be present are got rid of. (2) Strong, quickly-growing varieties of cabbage which have shown themselves to be resistant to the disease should be chosen.

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Phoma Napobrassicæ Rostr. appears on swedes (*Brassica Napus rapifera*) about the end of July or beginning of August and produces a dry rot on parts of the root. The attack starts from the outside, either near the neck of the root or sometimes at the side or at the apex of the root and spreads gradually. The infected cell tissues assume a light brown colour, later brownish-black, and the cells shrivel. Deep cracks arise and may lead to the formation of larger or smaller cavities within the root. The diseased patches are permeated by a mycelium which produces small, black pycnidia. Badly diseased roots undergo dry rot while still in the ground ; on stored roots that are only slightly affected the disease may spread further. Certain strains of swedes are especially susceptible to the disease which, it is supposed, is spread through the seed. This disease was first observed in Denmark in 1892, in 1911 in Norway, and there have been numerous individual cases in England, Ireland, and New Zealand (Rostrup, 1894 ; Hagen, 1912).

Phoma Chrysanthemi Vogl. sometimes attacks the leaves of greenhouse chrysanthemums and causes considerable damage. Brown to reddish-yellow, irregular spots, which originate at the margins of the leaves and spread inwards, appear, or else well-defined areas, grey at first and later becoming black, develop on the leaves. The flowers grow badly or die completely (Voglino, 1902). **Ph. apiicola** Kleb. causes the surface of the roots of celeriac to become brown in colour, scaly, and cracked. The internal tissues thus exposed are attacked by a number of insect pests and become filled with colonies of bacteria. When infected celeriac roots are kept in a damp storage place they usually develop a rot. Sometimes minute, globose pycnidia are found on the scaly portions of the roots or at the base of the leaves. From these pycnidia tendril-like conidia are extruded. Pycnidia containing viable conidia have been found in spring on the remains of dead roots. The disease also appears to be disseminated by seed coming from infected crops (Klebahn, 1910). **Ph. destructiva** Plowr. occurs on tomatoes, causing a peculiar darkening of the green tissues about the stalk of unripe fruits. The diseased area is depressed and later becomes covered with black pycnidia. The affected fruits begin to decay and fall prematurely before ripening (Brick, 1919).

Phoma pithya Sacc. appears to a more or less serious degree on the Douglas fir (*Pseudotsuga Douglasii*) as well as on other conifers such as *Pinus silvestris*, *P. montana*, *P. Strobus*, *Larix decidua*, etc. The previous year's shoots are seen to be constricted in places and on the sunken portions to be covered with a large number of small pustules, each of which contains a black, globose pycnidium. Above the constriction, new, healthy but weak shoots often grow out. Possibly the fungus is the conidial form of one of the Ascomycete fungi—*Diaporthe* (Saccardo, 1884 ; Rostrup, 1902 ; Petrak, 1923). **Ph. abietina** Hart. causes similar deformations on young and old specimens of white fir, *Picea alba*.

In the head of the tree, brown, dry twigs are found with black pycnidia on the depressed surface of the bark. Very closely related are **Ph. acicola** (Lév.) Sacc. on pine needles and **Ph. Pini** (Desm.) Sacc. on spruce needles; **Ph. alternariaceum** Brooks and Searte and **Ph. Lycopersici,** conidial form of *Didymella Lycopersici* Kleb. (Klebahn, 1921).

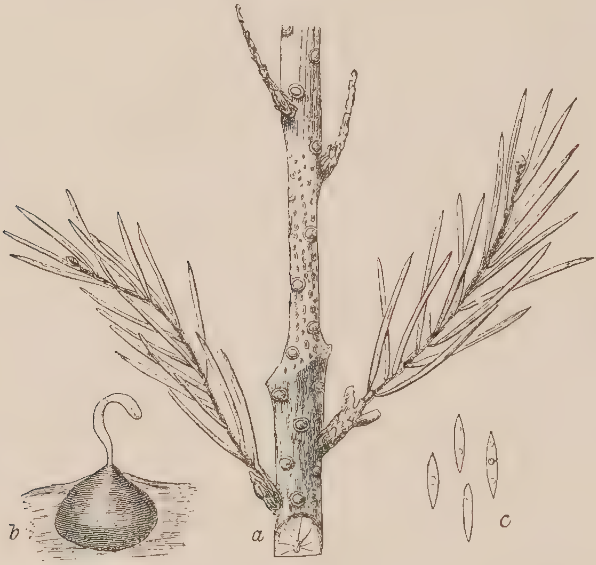

Fig. 351.—*Phoma pithya.*
a, portion of branch of *Pseudotsuga Douglasii* showing constriction and pycnidia; *b*, pycnidium; *c*, conidia. (After E. Rostrup.)

Of the species of *Phoma* found on deciduous trees the following may be mentioned: **Phoma fagina** Rostr. causes constrictions on the stems of beech seedlings. **Ph. quercella** Sacc. & Roum. on young twigs of oak. **Ph. sordida** Sacc. causes the wilting of shoots of hornbeam. **Ph. endogena** Vogl. & Bon. on the edible chestnut. **Ph. oblonga** Des. on young elm plants. **Ph. flaccida** Rev. & Verg. occurs on vines in French greenhouses. Black, shining streaks appear on the internodes of the stem. The affected stems are brittle and easily broken (Ravaz and Verge, 1925).

Septoria

The pycnidia contain long, slender conidia which are often guttulate or septate.

This genus also contains a large number of different forms of which the following may be mentioned: **Septoria Tritici** Desm. produces pale spots with small, black dots arranged in rows on the leaves of wheat, rye, and *Poa pratensis.* The fungus sometimes causes severe injury to young wheat seedlings in early spring (Eriksson, 1890; Weber, 1922). **S. Secalis** Prill. & Del. forms small, circular dots or larger, spreading patches on the leaves of rye, and pycnidia develop on the lesions. This form has been observed in France and in North America. Other forms found on *Gramineæ* are **S. Passerini** Sacc. on barley, **S. Agropyri** Ell. & Ev. on couch grass, **S. Bromi** Sacc. on *Bromus inermis* and other species, **S. Pastinacæ** West. on parsnips, **S. leguminum** Desm. on peas and beans, **S. Cannabinæ** (Lasch.) Sacc. on hemp, **S. piricola** Desm. on pears, **S. Rosæ** Desm. on roses, **S. Hydrangeæ** Bizz. on *Hydrangea hortensis*, **S. chrysanthemella** Sacc. on cultivated chrysanthemums, **S. Dianthi** Desm. on garden carnations, and **S. Azaleæ** on *Azalea indica*.

The following forms are important and require more notice: **Septoria ampelina** Berk. & Curt. causes a disease known as "Melanose" on the leaves of the vine. Small, brown spots, which finally become black and sometimes coalesce are produced. Pycnidia develop on both sides of the leaf. The pycnidia when ripe extrude large quantities of filiform, septate spores which quickly disseminate the disease (Viala, 1887). **S. Apii** (Br. & Cav.) Rostr. produces pale, light- to greyish-brown, sharply limited spots on the leaves of celery. The spots are covered on both sides of the leaf with abundant, punctiform pycnidia which occur also on the leaf-stalks and fruits. The opening of the pycnidium is drawn out to a short, dark-coloured papilla. In severe outbreaks of the disease many of the leaves fall. The disease has been known since the beginning of the present century and in some years causes much damage. The seed should be disinfected with Bordeaux mixture and the spraying

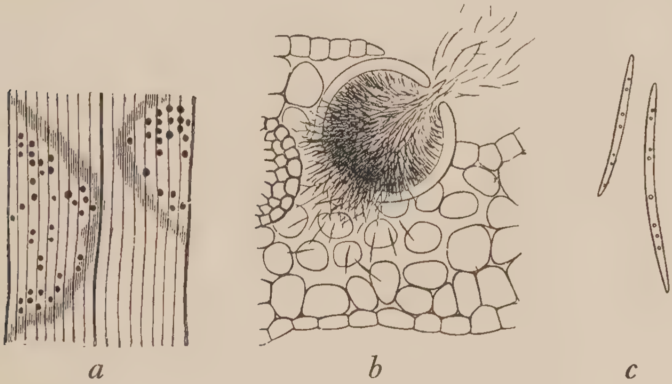

FIG. 352.—*Septoria Tritici* on the Wheat Plant.

a, portion of leaf with spots of fungus; *b*, section of a spot showing pycnidium; *c*, conidia. (Orig.)

or dusting of the plants with the same fungicide is also recommended (Halsted, 1891 ; Klebahn, 1910).

Pyrenochæta pubescens Rostr. gives rise to round to oval, dark, sunken spots on the stems and twigs of young trees of *Tilia grandifolia* and *T. parvifolia.* The spots are dark red at first, then whitish-grey, and finally blackish-grey and bear abundant small, blackish pycnidia. The pycnidia bear short, colourless, three-septate hairs on the upper surface. Numerous conidia, oval to elliptical in shape, are contained in the pycnidia. The affected twigs suffer severely and many buds are arrested in their development. If the spots extend and finally girdle the twig or branch the portion above is killed. Different species of lime trees vary in the degree to which they are attacked by the fungus. The disease was observed in nurseries in Denmark and Sweden. When it is noticed at an early stage on the twigs they should be cut off and burnt (Rostrup, 1902).

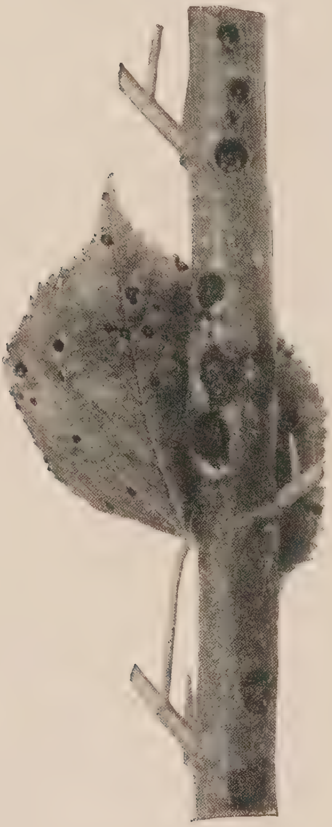

FIG. 353.—*Pyrenochœta pubescens.* Branch and leaf of lime with diseased areas. (Orig.)

Ascochyta

The pycnidia appear on stems, leaves, and fruits. The conidia are two-celled, colourless.

These fungi produce yellowish-brown spots on the organs that are attacked ; brownish-black, punctiform pycnidia occur in the middle part of the spot.

The following species belong to this genus : **Ascochyta Phaseolorum** Sacc. & Speg. and **A. Boltshauseri** Sacc. on beans, **A. Humuli** Sacc. & Speg. on hops, **A. Chrysanthemi** Stev. on cultivated chrysanthemums, **A. Apii** Halst. on celery.

Ascochyta piniperda Lindau causes the browning of the needles of spruce shoots, particularly those of young plants. After a short time the needles fall and the side branches hang down limply. Finally, the shoots dry up and the head of the plant may even die. Pycnidia appear at the base of the diseased shoots. The conidia are spindle-shaped, two-celled and are extruded in tendrils (R. Hartig, 1890, 1900; Lindau, 1900; Graves, 1914).

Other species of *Ascochyta* produce spots on the leaves of various trees and woody shrubs, *e.g.* **Ascochyta Tremulæ** v. Thüm on *Populus Tremula*, **A. Periclymeni** v. Thüm on *Lonicera periclymenum*, **A. Viburni** Sacc. on *Viburnum opulus*, **A. Syringæ** Bres. on *Syringa vulgaris*, etc.

In North America, **Sphæropsis Malorum** Berl. & Vogl. causes "canker" on the branches and stems of apple trees. The attack of the fungus occurs in the spring, the bark becomes discoloured, dry, and cracked. The development of the fungus ceases in August with the formation of conidial pustules. The fungus also attacks pears, quinces, and numerous species of the genera *Prunus*, *Rhus*, *Celastrus*, *Diosporus*, etc. Recently the disease has also been observed in France (Paddock, 1899; F. A. Wolf, 1913).

Diplodina Castaneæ Prill. & Del. known in France as "La Javart des Chatâigniers." Elongated, brown, shrivelled patches occur on young shoots of the chestnut. These patches crack, form deep fissures, and finally true cankers, which continue to enlarge and may after some years cause the branch or stem to die. Sometimes small, flattened, simple or divided pycnidia occur in the cankers. The conidia are spool-shaped, hyaline, and transversely septate. The woody tissue of the affected branch often dies in the first year. After the cankers have continued to enlarge for some years the trees themselves are killed. This disease was discovered in 1893 in France and described; some years later it was observed in Brittany.

As a control measure it is recommended to destroy all infected plants as well as surrounding healthy ones and to plant other species of trees (oak, beech) in their place. Mixed stands of trees are preferable in this case.

CHAPTER XVII

MELANCONIACEÆ

The conidial stromata are flat and without any special receptacle.

Glœosporium

The stromata are flat and disk-shaped and they become visible after the cuticle has burst open; they are often of a yellowish or brownish colour. The conidia are hyaline, ellipsoidal, one-celled, and are extruded as salmon-coloured gelatinous masses. In certain species, which are often distinguished by the genus name *Colletotrichum*, long or short hyphæ (paraphyses) are found within the circumference of the stroma.

The fungi belonging to this genus produce discoloured patches of varying form and size on the leaves and younger parts of the stem (annual shoots). They cause premature withering and leaf-fall. The diseases are known in general as "Anthracnoses."

Anthracnose or Scorch of Clover

Glœosporium caulivorum Kirchn.

This disease was first observed in America and later in many parts of Europe. It attacks red clover and sometimes also alsike clover. Elongated, brown patches, deeply sunken in the centre and with a broad black margin, arise on the stems and leaf-stalks. The leaves above the affected parts often wilt. Conidial stromata develop on the sunken portions of the lesions. The disease has, in some cases, destroyed from 25–50 per cent. of the crop; American clover suffers most severely.

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A closely related species **Glœosporium Trifolii** Peck. only attacks the leaves of clover. Other species belonging to this same genus are: **G. graminum** Rostr. with numerous small, brown stromata on the leaves of rye grass, and **G. Dactylidis** Rostr. on cocksfoot grass; brown wart-like pustules appear on the upper branches of the panicles. Both of the above-mentioned species have been observed in Denmark.

Marssonia Secalis (Oud.) Magn. produces elongated, greyish-white patches with brown edges on the leaves of rye and barley as well as on a number of wild grasses. On the under side of these patches, small stromata arise; the conidia are divided transversely.

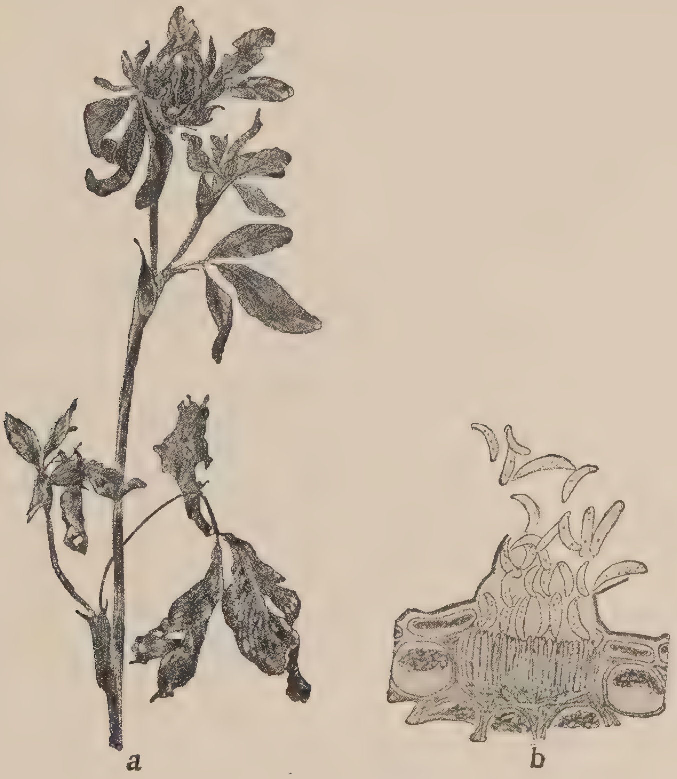

FIG. 354.—Anthracnose of Clover.
a, red clover plant with elongated dark spots on the stem; *b*, section of stroma with conidia. (After O. Kirchner.)

Cryptosporium leptostromiforme Kühn causes the formation of light-coloured patches, which later become brown, on the stems of yellow and blue lupines. Elongated black conidia appear on the lesions. Plants which are severely attacked die prematurely, often before flowering. The fungus can continue to live in the ground on the lupine stems. Where the soil is known to be infected, lupines should not be grown for two or three years.

Chromosporium Maydis (Ces.) Sacc. causes green spots or rings on the surface of maize grains; the fungus is found in France and Italy.

Anthracnose of Vine

Glœosporium ampelophagum (de By.) Sacc.

This disease attacks all parts of the vine above ground. The fungus which causes the disease has been given other names—*Sphaceloma ampelinum* de By., *Ramularia ampelophaga* Pass., and *Mangiana ampelophaga* Viala & Pac. The disease is characterized by brown, black-bordered, sunken spots on the stem-parts, leaves, and berries. In the beginning, the spots are very small, hardly as big as a pin's head; those on the stem-parts enlarge lengthwise and coalesce to form long, reddish, canker-like lesions. Young shoots often die back completely; on older shoots the fungus penetrates to the tissues of the wood and medulla and destroys them. The vines finally become black, and their appearance suggests that they have been destroyed by fire. On the leaves and berries the spots remain circular or oval, each spot being surrounded by a black ring. Badly affected leaves curl and become of a reddish colour, and in this state they fall prematurely. The injury to the grapes is

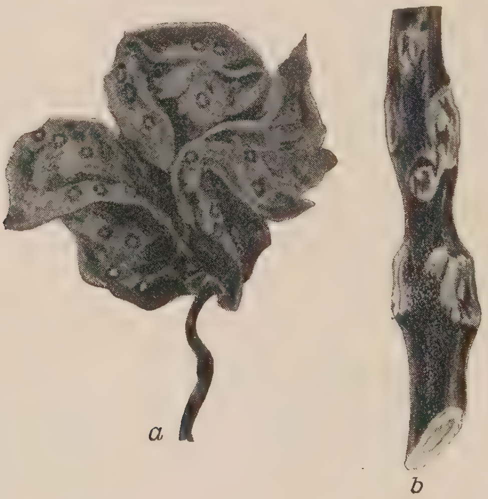

Fig. 355.—Anthracnose of Vine.
a, severely injured leaf; *b*, portion of stem attacked by fungus.
(After G. Linhart and G. Mezey.)

generally limited to a spotting of the skin; the inside develops normally and remains juicy.

Warm, damp weather, especially in spring, and planting the vines too closely favour the development and spread of the disease.

The fungus fruits from May to September in the lesions on the twigs and also in the spots on the leaves and berries. A dense superficial stroma is formed from which parallel conidiophores arise. The conidia are minute, one-celled, hyaline (microconidia), and they can germinate at once. In addition, sunken pycnidia are found in the underlying tissues of the diseased spots; they are surrounded by rhizomorph-like hyphæ and develop larger conidia (macroconidia).

In artificial cultures other developmental forms of the fungus are said to have been distinguished; in one case yeast-like cells which germinate with the formation of spores or by hyphæ, in another chlamydospores arranged in chains.

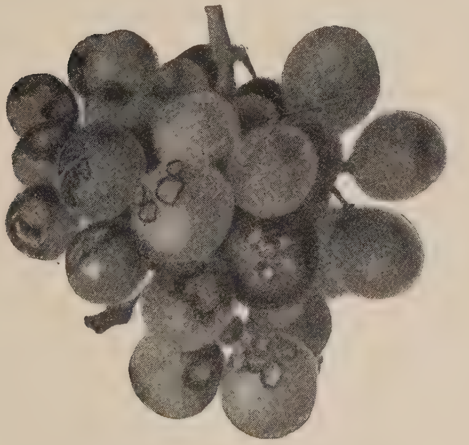

Fig. 356.—Anthracnose of Vine. (After G. Linhart and G. Mezey.)

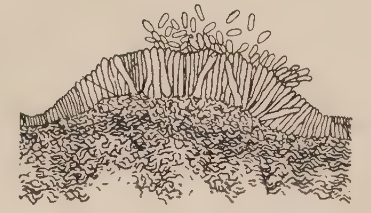

Fig. 357.—Anthracnose of Vine. Conidial pustule. (After P. Viala.)

This disease has been known from earliest times in the vine-growing countries of the Old and the New World and it has sometimes been very destructive.

Control Measures:

(1) The vines should be dusted in summer, first with finely-divided sulphur and in later applications with sulphur to which $\frac{1}{5}$–$\frac{3}{5}$ of powdered lime has been added. (2) In winter, the vines should be thoroughly sprayed with an acid solution of sulphate of iron (110 lbs. crystallised ferrous sulphate, 1 qt. sulphuric acid, 22 gall. water).

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Anthracnose of Runner Beans

Colletotrichum Lindemuthianum Sacc. & Magn.

This disease appears on the pods, sometimes also on the stems and leaves, of all kinds of dwarf and runner beans. On the young, green pods the spots are at first small, circular, brown with purple margin; later they are elongated, confluent, and sunken in the centre. The spore-masses arise as black dots on the depressed spots on the pods. If the attack is an early one, the spots penetrate the wall of the pod and infect the seeds or even the cotyledons within the seed coat. When such infected seeds germinate, the cotyledons first become diseased, then the stem and leaves, and finally the pods.

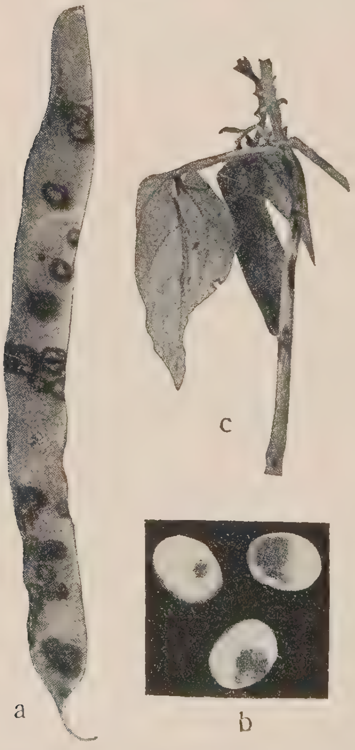

Fig. 358.—Anthracnose of Runner Bean.

a, diseased pod; *b*, diseased beans; *c*, stem and leaves attacked by fungus. (Orig.)

The first thorough investigation of this disease led to the view that it was carried in the seed (Frank, 1883). The mycelium of the fungus was thought to pass from the disease spots on the fruit walls to the underlying seed and to penetrate the seed coat and cotyledons. By means of the seedlings produced from such infected seeds the disease was thus carried over to the new crop. This view is not, however, supported by more recent observations made in America and Europe.

The exhaustive experiments carried out in the years 1906–21 at the New York Experiment Station, Ithaca (Whetzel, 1906–07; Barrus, 1911–21), showed that in the seed beans themselves an internal source of infection is to be sought. More recent investigations undertaken in Sweden

(Eriksson, 1924) also lead to the same conclusion. The form in which the internal disease-germ is present has not yet, it is true, been made clear. The possibility exists that in seeds, although apparently quite healthy, harvested from diseased plants the fungus may be united symbiotically with the protoplasm of the host cells to form a " mycoplasm-symbiosis," as all attempts to discover other fungous elements, such as spores or mycelium, have failed.

The investigations in America have led to different strains of the fungus being distinguished. At the end of 1918, two such strains—alpha and beta—were known. The majority of the beans which were examined —more than 300 in number—showed themselves to be susceptible to one or other of the strains of the fungus, while some were susceptible to both strains. Only a few sorts of beans were resistant to both strains of the fungus, the most important of these being Wells Red Kidney and White Imperial. Wells Red Kidney was immune to the alpha strain and very resistant to the beta strain, while White Imperial was resistant to the alpha strain and practically immune to the beta strain. Latterly, a third strain—gamma—has been found on a crop of White Imperial (Burkholder, 1923). It has been shown by artificial infection experiments that Wells Red Kidney is very susceptible to the gamma strain of the fungus and only one variety of bean—Michigan Robust—is resistant.

The Swedish infection experiments showed that the causal organism found in the pustules which break out on the surface of the pods was only able, by means of its conidia, to infect healthy plants if they were not at a greater distance than 5–10 metres from the source of infection.

The very complete study of the disease which has recently been made in Germany (Schaffnit and Böning) has thrown much light upon the nature of the fungus and the conditions under which it lives. It is now hoped that means of controlling this pest will be found.

This disease was first investigated scientifically in 1883 in Germany by Frank. In 1892 it was described from the Ithaca Experiment Station, America (Beach), as being the cause of much loss, and during the first two decades of the present century it was regarded as the most serious bean disease in North America. In Sweden it has been known since 1910, and during the three following years it spread widely in that country as well as in other parts of Europe.

Control Measures :

(1) Seed should only be taken from healthy crops. (2) All diseased parts of plants should be burnt. (3) Beans should not be grown for a few years on ground where the disease has occurred.

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Anthracnose of Cucurbitaceæ

Colletotrichum lagenarium (Pass.) Ell. & Halst.

This disease attacks cucumbers grown in glasshouses, in frames on hot-beds, and in the open. It is indicated by a zone of pallid tissue on the margins of the leaf and by the presence of a large number of scattered

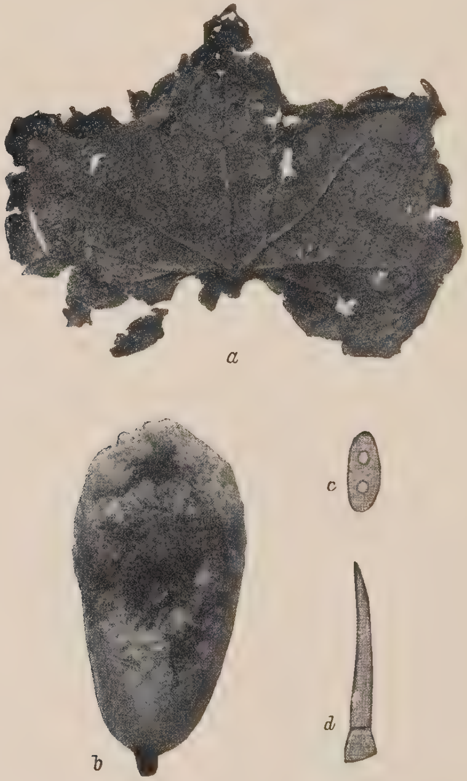

Fig. 359.—Anthracnose of Cucurbitaceæ.
a, diseased leaf of cucumber; *b*, diseased cucumber; *c*, conidium; *d*, paraphysis. (Orig.)

spots of irregular shape on the lamina. The affected parts of the leaf soon die and drop out, which gives a torn, ragged appearance to the lamina. Elongated, greyish-white streaks are formed on the stems. The set of fruit is very small and the fruits that develop are seldom longer than 3 cm. The disease appears, mainly, at the tip of the fruit in the form of a large, pale spot on which, particularly in the centre, are close, salmon- or brick-red felts of mycelium. From the hyphæ numerous elongated conidia are separated. On the edges of the pustules are a few elongated, pointed paraphyses. Growers generally consider that the disease is spread by the seed.

It is stated that the fungus is able to live over the winter but not for a period of twenty months; consequently a two-years' rotation ought to prevent infection of the soil (Gardner and Gilbert, 1921).

The disease was first observed in 1867 in Italy (Padua) on pumpkins, and now appears to be found in all countries where cucurbitaceous plants are grown; in many places it has led to the complete destruction of the crop.

Control Measures:

(1) Only such seed as has been obtained from a disease-free crop should be sown. (2) Cucumbers should not be grown in the same soil for two successive years. (3) The crops should not be hoed except in dry, sunny weather. (4) In glass houses, all diseased parts of the plants should be destroyed and the houses (walls, windows, stages, and floor) should be carefully cleansed and treated with some fungicide such as 2 per cent. Bordeaux mixture, milk of lime, etc. The beds should be made up with fresh soil and during the growing season the atmosphere should be kept moderately damp. (5) The crops should be sprayed repeatedly with Bordeaux mixture in June and the first half of July.

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Other anthracnose diseases which occur on horticultural crops are: **Glœosporium phomodes** Sacc., which forms small, black, raised spots about the size of a pin's head on the fruits of the tomata plant; the pycnidia are embedded in a stroma-like tissue. Coloured, irregularly-shaped chlamydospores, which are able to germinate at once, are also found. Infection of the fruit takes place through wounds in the skin. The disease has appeared in Western Europe and North America (Guéguen, 1902). **Gl. (Colletotrichum) Althææ** Southw. produces brown spots on the leaves of some species of mallow and causes the leaves to wither. Sometimes sunken, yellowish-brown to black spots occur on the leaf-stalks and on the stems. The disease has, in some cases, been so destructive that great difficulty has been experienced in growing the mallows (Southworth, 1890). **Gl. (Colletotrichum) oligochæton** Cav. gives rise to spots, carrying tufts of salmon-pink conidia, on the leaves, stems, and fruits of pumpkins, gourds, and melons. When the fungus attacks the stem, the parts above the lesion are killed. The fruits do not ripen, but decay and fall from the plant. The fungus has been very destructive to gourds in Italy (Cavara, 1889) and to melons in France (Prillieux and Delacroix, 1894), and is at present regarded as the most serious parasite on the leaves of the cucumber in England (Bewley, 1922). **Gl. filicinum** Rostr. produces brown spots with white conidial pustules on the leaves of *Aspidium Filix mas*. The foliage becomes stunted. **Gl. Strutiopteridis** Rostr. The distorted foliage is covered with white conidial cushions and wilts prematurely. **Gl. olivarum** d'Alm. This fungus is found on the ripe or ripening fruits of the olive tree. Well-defined, almost circular, depressions occur on which small pycnidia grow out and in damp weather extrude a slimy mass of conidia which are of a yellowish colour at first, then brown. Sometimes the fruits dry up before the pycnidia have opened and become wrinkled and brown ; many of the fruits drop. The disease does not attack all the trees in the garden, but when a tree has become infected the disease reappears regularly each year ; it is known under the names of " Gaffa " or " Lebba " in Portugal (d'Almeida, 1899).

A number of anthracnose diseases have been found on plants growing in the greenhouse. **Glœosporium elasticæ** Cke. & Mass. causes yellowish-brown spots on the leaves of *Ficus elastica*. **Gl. cinetum** Berk., large, greyish-brown, zebra-like transverse bands on the leaves of species of *Cattleya*, *Stanhopea*, and *Dendrobium*. **Gl. affinis** Sacc. : brown to black spots are produced on the leaves, leaf sheaths, and stems of *Cœlogyne*, *Cattleya*, etc. **Gl. Oncidii** Sacc. white, semiglobular conidial layers on the leaf sheaths of *Maxillaria infestans*.

Of the anthracnose diseases which occur on foliage trees in parks, etc., the following may be mentioned : **Glœosporium acerinum** West. on maple ; irregular discoloured spots of varying size on the leaves. **Gl. quercinum** West. on oak ; brown to brownish-yellow spots with conidial pustules on the under surface of the leaves. From these pustules, short, yellow tendrils of conidia are extruded. **Gl. fagicolum** Pass. on beech ;

spots similar to the last-mentioned; premature fall of leaves (Morstatt, 1909). **Gl. inconspicuum** Cav. on elm; ochre-yellow circular spots on the leaves. **Gl. Salicis** West. on willow; numerous small, dark brown, shining leaf-spots with white conidial tendrils; sometimes almost all the leaves of a tree are affected. **Gl. Tremulæ** Lib. on poplar; large grey leaf-spots with numerous dark conidial pustules arranged in concentric rings. **Gl. amygdalinum** Brizi on almond; yellowish-brown vesicles on the young fruits, to a less degree on the young twigs. The shining, soft, hairy growth on the fruit disappears and a wound-like depression arises in the fruit wall. The fruit decays or withers according to the weather (Brizi, 1896).

Melanconium fuliginosum Scrib. & Viala. This disease is known as "Bitter Rot" in America and appears on ripening grapes. The skin of the berry becomes wrinkled, and below the skin dark conidial layers are formed which pour out their contents as bright red tendrils. The berries shrivel and remain hanging on the vine. In abnormally damp weather the pedicels and young shoots are attacked by the fungus; in such cases the berries become plump and turgescent and do not dry up until later (Noack, 1899).

Cylindrosporium Padi Karst. attacks different species of the genus *Prunus*, in Europe especially *Prunus Padus* and in North America *P. cerasus*, *P. domestica*, *P. armeniaca*, and *P. serotina*. In Europe the fungus causes much damage to nursery seedlings of *Prunus Padus*. The lesions are on the under surface of the leaf in the form of small conidial pustules. The conidia are filiform, slightly bent, usually one-celled, sometimes divided by false septa, and they germinate easily. Leaves that are severely attacked curl upwards and soon die. Large beds of seedlings may be destroyed in this manner (Stockholm, 1884).

In North America the fungus attack on species of *Prunus* takes place about the middle or end of May in the case of young trees and somewhat later on older ones. A number of scattered, pale red spots, small at first and gradually becoming larger and confluent, occur on the upper surface of the leaf and spread until the leaf finally falls. Sometimes the spots, when they have increased in size, are torn open and lead to a perforated or "shot-hole" condition of the leaves. Cherry seedlings in the nursery are often very severely attacked by this fungus in America (Pammel, 1891; v. Höhnel, 1924).

This fungus was first described from Finland in 1885; since 1891 it has been known in North America as one of the most stubborn pests met with in the growing of cherries and plums (Fairchild, 1893). As different sorts of cherries and plums vary in their resistance to the fungus, this property has been made use of in America with good results in controlling the disease.

Cylindrosporium castanicolum (Desm.) Berl. causes the leaves and fruits of the edible chestnut to wither and dry up. Small brown, later black, spots appear on the under surface of the leaves, which roll together spirally and wither. Frequently the fruits are also attacked and the spines and parts of the fruit wall turn red, later black. On the spots, the spore-masses are produced in covered pustules. The spores are filiform, hyaline, three- to four-celled. Fallen leaves should be collected and burnt. In 1893 the chestnut crop in mid-Italy was almost completely destroyed (Berlese, 1893).

To this same genus belong also **Cylindrosporium Tubeufianum** All., which attacks the fruits of the bird cherry (v. Tubeuf, 1895) and **C. Juglandis** Wolf. on the leaves of the walnut (*Juglans regia*). The latter disease has appeared to a devastating degree in Alabama, North America (Wolf, 1914).

Pestalozzia

Disk- or cushion-shaped spore-layers, black, laid bare by the bursting of the epidermis. Spores spindle-shaped to elongated, greyish-brown, except the small, colourless apical cells, three- or many-celled, with one or more hyaline setæ on the apical cell.

Pestalozzia Hartigii Tul. attacks young one- to three-year-old plants of coniferous and deciduous trees. The fungus causes a characteristic constriction of the stem just above ground-level. Above the constricted area there is a hypertrophied swelling of the stem. The attack of the fungus has the same effect as the " ringing " of the bark, the foliage turns yellow and the plant dies. In winter, if the dead stem is covered with a layer of moist, withered leaves, pycnidial cushions arise which are white at first and later become black. The pycnidia burst when filled with conidia. The disease has been observed on fir (especially *Abies pectinata*), pine, copper beech, oak, alder, maple, etc. To control the disease it is recommended to burn all the diseased plants, and in the case of a severe outbreak to dig over the soil (v. Tubeuf, 1888 ; Rostrup, 1891, 1893 ; Lagerberg, 1911).

Many other fungous plant pests, both out of doors and in the greenhouse, belong to this same genus, and the following may be mentioned : **Pestalozzia funerea** Desm. attacks a large number of Gymnosperms, such as species of the genera *Cupressus*, *Biota*, *Thuja*, *Cryptomeria*, *Retinospora*, *Salisburia*, *Chamæcyparis*, *Juniperus*, etc., producing annular constrictions in consequence of which the needles become yellow and later brown. **P. uvicola** Speg. gives rise to yellow spots on the leaves and berries of the vine. **P. compta** Sacc. produces irregular, yellow spots with red margins on the upper surface of the leaves of cultivated roses. **P. Coryli** Rostr. causes numerous brown spots on fresh, green leaves of hazel. **P. maculicola** Rostr. produces numerous small, round, straw-coloured spots with brown margins on the leaves of *Ulmus montana*. **P. Lupini** Sor. causes rust-red spots on the cotyledons and leaf-tips of *Lupinus mutabilis* and *L. Cruickshanksii.* The plants die off. **P. fuscescens** Sor. imparts a general grey, almost milky appearance to *Corypha australis*; on individual leaves pale, translucent spots with black margins, and at the base of the petioles saucer-shaped depressions with black, punctiform spore-layers. At the same time a diseased condition of the roots is clearly observable. **P. Guepini** Desm. gives rise to large, light-coloured spots with dark margins on the leathery leaves of a number of greenhouse plants, such as *Camellia*, *Citrus*, *Magnolia*, *Rhododendron*, *Thea*, etc. The leaves fall prematurely.

Septoglœum Hartigianum Sacc. causes the death of young maple twigs. In spring the young shoots are killed whilst the lower branches remain green. The mycelium, which is present in the diseased bark, breaks through the epidermis and develops small, pale conidial-masses in long, greyish-green lines. The early, May shoots which are infected by the conidia do not die until the following year. The disease is widely spread in Germany and Switzerland (Hartig, 1892). **S. Mori** Briosi & Cav., known in Italy as "Forsa," gives rise to yellow spots with brown margins on the leaves of the mulberry tree. On these spots conidial layers are formed beneath the epidermis. In autumn the spore form known as *Pleospora moricola* Pass. appears. The infected leaves are not eaten by silkworms. The fungus can over-winter on the leaf-stalks and young twigs. Warm, damp conditions are favourable to the development of the fungus. **S. saliciperdum** Tub. & All. causes the death of the twigs of willows (*S. pentandra*, etc.) and poplars (*Populus tremula* and *P. alba* (v. Tubeuf, 1902).

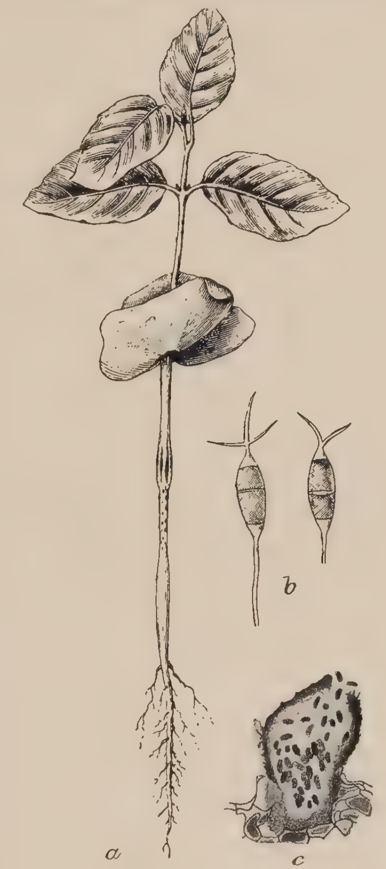

FIG. 360.—*Pestalozzia Hartigii.* *a*, diseased beech seedling; *b*, conidia; *c*, pseudopycnidium. (*a* and *b*, after E. Rostrup; *c*, after T. Lagerberg.)

CHAPTER XVIII

HYPHOMYCETES

The conidiophores are formed singly; there is no surrounding envelope (periderm).

Ramularia

The conidiophores emerge in small, white cushions from the stomata on the under surface of small, round leaf-spots which are of a pale colour on the upper surface. The fungus attacks a number of different species of plants. The conidia are cylindrical, 1–3 septate.

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Ramularia Betæ Rostr. produces circular, greyish-white spots on the leaves of beet, mangel, etc. The spots are surrounded by a brown margin, and the white appearance of the centre is due to the hyphæ and conidia. At times the disease has been so severe in Denmark that almost every leaf in the field has been affected to a greater or less degree.

Similar leaf-spots are caused by **R. sphæroidea** Sacc. on vetches, **R. Onobrychidis** Allesch. on sainfoin, and **R. Schultzeri** Bäuml. on bird'sfoot trefoil. The closely-related *Ovularia deusta* Sacc. forms small, pallid spots, which bear tufts of hyphæ, on the leaves of the chickling vetch.

Ramularia Spinaciæ Nyp. gives rise to spots, pale on the upper surface and brown on the lower, on the leaves of spinach. **R. Cynaræ** Sacc. on artichokes in France, develops numerous, roundish spots covered with a white felt. The spots are confluent and finally cause the death of the leaf. The heads of the artichokes wither. **R. Armoraciæ** Fuck. on horseradish, causes circular, brownish, later white, leaf-spots, on the under side of which is a white covering of conidial tufts; the leaf dies prematurely and the root is checked in its development. The conidial stage appears to be one of the species of *Entyloma* belonging to the *Ustilaginaceæ* (Marchal and Sternon, 1924). **R. lactea** (Desm.) Sacc. forms white spots with brown margins on the leaves of several species of violet; the production of flowers is reduced. This disease appeared very severely in 1922 in Holland; it can be controlled by spraying with Burgundy mixture (v. Keulen, 1924). **R. variabilis** Fuck. occurs on the leaves of species of *Digitalis*, and appears to be the conidial form of a species of *Entyloma* belonging to the *Ustilaginaceæ* (Marchal and Sternon, 1924; Killian, 1926).

Cycloconium oleaginum Cast. is found in late summer and autumn on the leaves of the olive tree. Noticeable roundish spots, more or less numerous, at first blackish- and finally greyish-white with black margins; chiefly on the upper surface of the leaf. The mycelium in the epidermal cells sends out short branches which break through the cuticle and produce conidiophores. The conidia are the cause of the noticeable blackish colour which the spots have at first. The infected leaves fall prematurely; in severe attacks the whole tree may be defoliated. More rarely the pedicels and the fruits are attacked, with the result that the latter drop before becoming ripe (Brizi, 1895; Prillieux, 1897; Ducomet, 1907; Petri, 1913).

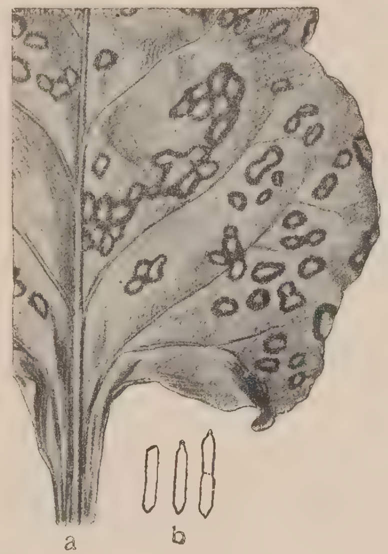

FIG. 361.—*Ramularia Betæ.*
a, portion of leaf with spots of fungus; *b*, conidia. (After E. Rostrup.)

The various cultivated sorts of olive show a marked difference in the degree to which they are affected. In Italy the following sorts are regarded as very resistant to the disease: Leccina, Elcina, Olivago, Silvestrone, Carboncella, and Cellina o Nardo. The very susceptible varieties in Italy are Taggiasca, Martellina, Marinella, Pendula, Frantuana, Grossaia, etc., and Lucques, Amellan, Rouget, and Verdale in France.

Control Measures:

(1) Only those varieties that have proved themselves locally to be resistant to the disease should be planted. (2) The soil should be manured to remedy an eventual lack of lime and to assist the development of a healthy, active root system. (3) Spraying with Bordeaux

mixture at the proper time, in dry districts in January or February, and in very wet districts once in spring and once in autumn (Petri). Olive gardens on low ground should be sprayed for the first time in August, then in November or December, and a third time from February to March. Where the olive trees are grown on high ground they should be sprayed first in September and once again in January or February (Ducomet).

Trichothecium roseum (Bull.) Link. produces a "Bitter Rot" of the fruits of plum, apple, pear, cherry, orange, melon, etc. The internal mycelium causes the flesh of the fruit to decay and to become tough and acquire an intensely bitter taste. In damp places a white mould develops on the fructifications of the fungus, which are often of a reddish colour. When a diseased plum is cut across it is seen that the normal, yellowish olive-green parenchyma has become browned and is traversed by reddish streaks radiating from the stone layer. The mycelium is colourless and much branched. In the moist chamber a whitish mould, which later becomes pink, is developed. A single, pear-shaped, two-celled conidium is abstricted at the top of the conidiophore, or if the food supply is abundant a head of conidia may be formed. The affected fruits are inedible. Similar lesions due to the same fungus have been met with on hazel nuts (*Corylus Avellana*) and on the seeds of *Pinus Cembra*. The nuts show outwardly no sign of disease, but when opened a pink mould is found on the inner wall of the shell and on the kernel; the latter is inedible (Iwanoff, 1904).

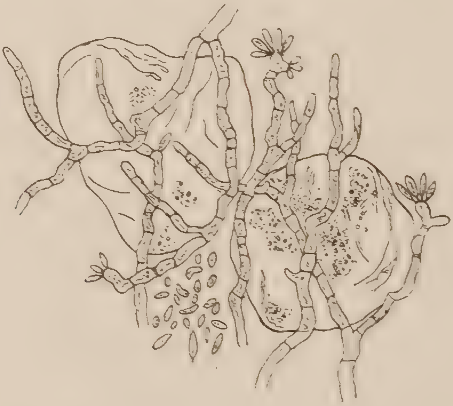

FIG. 362.—*Phymatotrichum baccarum*. Tissue of berry with hyphæ. (After C. A. and A. Oudema.)

Closely related to the above fungus is **Phymatotrichum baccarum** Oud., which causes a similar "Bitter Rot" on gooseberries. Ochre-yellow spots, later becoming brown, are found on the ripening berries, mainly at the base. A richly branched, septate mycelium is found in and between the cells of infected berries which are beginning to swell. Certain of the hyphæ in the intercellular spaces enlarge at the tips and produce basidia which abstrict several (3–10) ellipsoid, colourless conidia. The diseased berries are inedible (Oudemans., 1900).

Cucumber Scab

Cladosporium cucumerinum Ell. & Arth.

This disease attacks cucumbers, melons, and pumpkins growing out of doors or in greenhouses and frames. The disease is first noticed on the young fruits in the form of a few or many sharply defined, grey to black depressed spots, the ground of which is covered with a close network of greyish-green to black hyphæ. From the tips of the hyphæ one-celled conidia are abstricted. Holes finally appear in the leaves, and the fruits become deformed, one side being shrivelled and cracked.

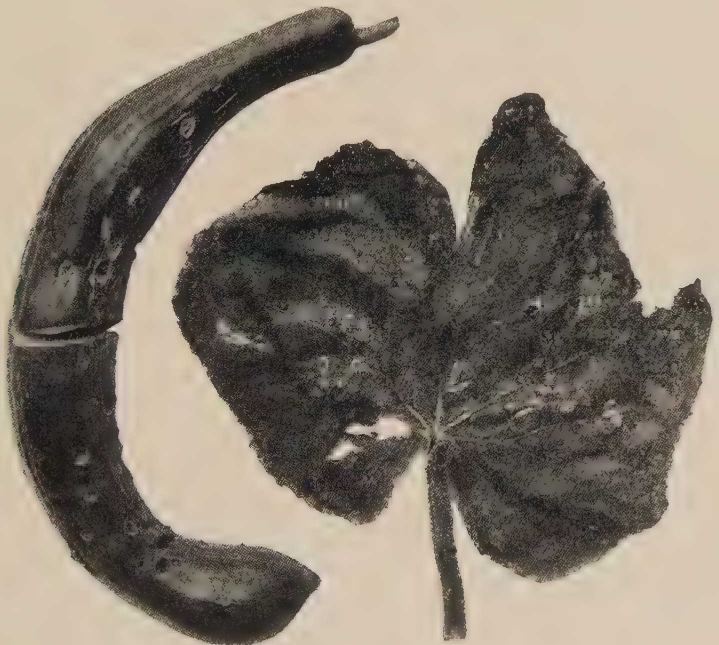

Fig. 363.—*Cladosporium cucumerinum.* (Orig.)

This disease was first noticed in 1889 in America, and in 1893 it was reported from Germany and England. Later, in 1905, it was found in

Norway and since 1907 in Sweden, where, in some years (1909–12), it has been widespread and the cause of much loss.

It is thought that the disease, like several other epidemics that occur in the cultivation of cucumbers, is to be attributed to the large scale on which the crop is grown (in England, for example, as many as a hundred large glasshouses may be found in one commercial garden) and to the very moist hot-house conditions. Also, that seed obtained from such crops carries the diseases from one place or county to others (Eriksson, 1915).

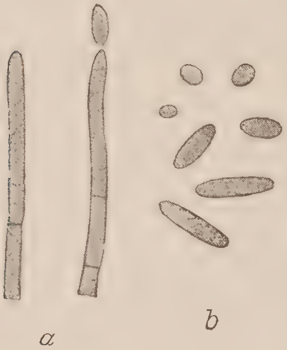

Fig. 364.—*Cladosporium cucumerinum.*

a, conidiophores ; *b*, conidia. (Orig.)

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Cladosporium fulvum Cooke produces yellow, later brown, spots in the angles of the veins of tomato leaves, especially in the glasshouse. On the under surface of the spots thick wefts of brown conidiophores are formed. The leaves, and finally the plants themselves, wilt. The formation of fruit is almost completely suppressed. The disease was first observed in America in 1883, in England (where it is known as Tomato Leaf Mould) in 1887, and it is now found in all European countries where tomatoes are grown. Good results have been obtained by spraying with Solbar (1 per cent.) each third week, combined with good ventilation of the glasshouses (Spangler, 1924 ; Hofferichter, 1924). Dusting with sulphur has also been recommended (Bewley).

Cerathophorum setosum Kirchn. produces dark brown spots on the leaves, leaf-stalks, and stems of young plants of *Cytisus Laburnum* and *C. capillatus*. The spots are visible on both sides of the leaf and are small at first, increasing later in size and soon covering the greater part of the lamina. The lower leaves are the first to be attacked and killed, afterwards the upper ones. A colourless, much-branched, septate mycelium is found within the infected organs, and on the surface punctiform, black tufts of conidiophores and conidia. The conidia are cylindric-fusoid and consist of 3–8 cells placed one above another. The middle cells are dirty-brown and the end cells of a lighter colour. The apical cell bears 3–5 long, straight, colourless bristles which are directed upwards or sideways. The conidia germinate easily and quickly. In addition to conidia the fungus also develops chains or knots of darker thick-walled chlamydospores which, after over-wintering in the open, germinate in the following spring (the beginning of June) and give rise to a fresh outbreak of disease (Kirchner, 1892; Rostrup, 1905).

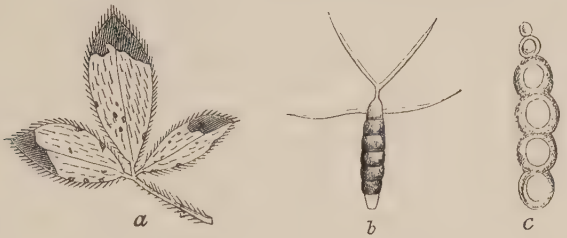

FIG. 365.—*Ceratophorum setosum.*
a, diseased leaf; *b*, conidium; *c*, chlamydospore. (After E. Rostrup.)

Heterosporium echinulatum (Berk.) Cke. causes serious damage to carnations and pinks both out of doors and under glass. Spots occur on the leaves, stems, and sepals, at first whitish, becoming blackish in the centre and finally black with white margins and covered with a network of brownish hyphæ. Conidiophores emerge from the stomata in tufts; the conidia are cylindrical with small spines and usually four-celled. Below the point of abstriction of a conidium, the hypha elongates, and after a short time develops another four-celled conidium; this may take place five or six times with the same hypha. In this way, the upper part of the hypha acquires a characteristic, knotted appearance. The mycelium spreads between the cells of the infected tissue without forming haustoria. Below the epidermis, sclerotia-like masses of hyphæ are produced. Diseased plants usually fail to flower and often die prematurely. Perithecia-like bodies have been found on the over-wintered remains of diseased carnation plants. To combat the pest in the greenhouse it is necessary to avoid a very damp, stagnant atmosphere (Rostrup, 1887; P. Magnus, 1888; Sorauer, 1898; Dowson, 1913).

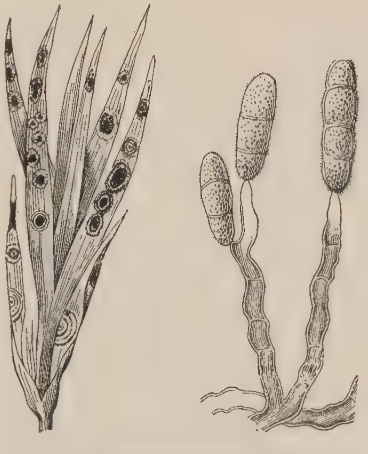

FIG. 366.—*Heterosporium echinulatum.* (After E. Rostrup.)

Heterosporium gracile (Wall.) Sacc. attacks the leaves of narcissus plants shortly after flowering. The leaves, which until then had been healthy and green, turn yellow, usually the edges first, and thereafter the whole leaf quickly becomes yellow and dry. Owing to the premature dying off of the foliage, the newly-formed bulbs are small and almost valueless. On the withered leaves, a dark coating of olive-brown conidiophores is found from which pale olive-brown, cylindrical conidia arise. The conidia are divided by several septa and covered externally with delicate spines which are often difficult to discern; the conidia germinate immediately. The different varieties of narcissus vary in the degree to which they are susceptible to this disease. At the beginning of the present century, the following Dutch sorts appeared to be immune: *Narcissus albo pleno odorato* (double), *N. poeticus ornatus* (single), all Leedsi and Stella varieties and all Jonquils, Campernelle Jonquils, and Polyanthus Narcissi. In some years the disease causes a large amount of damage in the large bulb-growing nurseries in Holland. As a means of controlling the disease it is recommended to spray the plants with Bordeaux mixture on the first appearance of the symptoms (Ritzema Bos, 1901, 1903).

Leaf Blotch of Cucumber and Melon

Corynespora Melonis (Cke.) Güss.

This disease appears on cucumbers and melons growing under glass. Small, scattered, translucent spots occur on the leaves. The upper side of the spot is sometimes covered with a thin layer of black, powdery mould. Similar spots also occur on the stems, but they are less numerous and of an oval shape. The fruits when attacked may show large, dark,

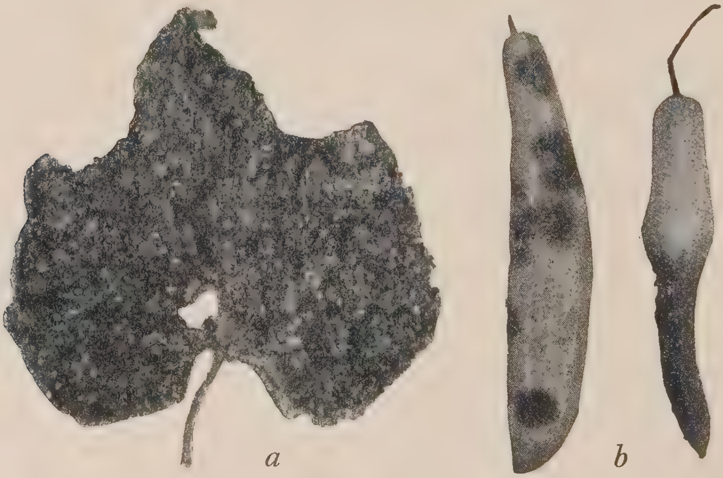

Fig. 367.—Leaf Blotch of Cucumber.
a, diseased leaf; *b*, diseased fruits. (Orig.)

somewhat depressed spots which crack and merge into the surrounding green tissue. In other cases the fruits become constricted or badly deformed at the tip or base.

The sooty coating found on the diseased fruits consists of greyish-brown, segmented hyphæ which pass through the stomata in groups. Bobbin-shaped, three- or four-celled conidia are produced from the tips of the hyphæ. The fungus thrives best in a very moist, stagnant atmosphere. The disease is spread not only by dried infected leaves, which under damp conditions develop conidia, but also by the packing boxes, etc., in which the fruit is sent from a centre where the disease occurs.

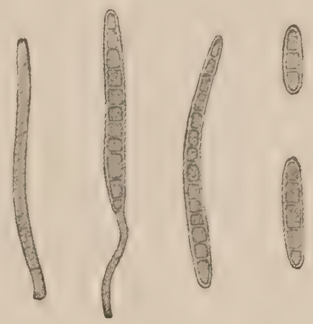

Fig. 368.—Leaf Blotch of Cucumber. Condiophores and conidia. (Orig.)

The disease can best be controlled by keeping the glasshouse dry, especially at night, or by removing and burning the infected leaves before they fall. Only seed from proved healthy plants should be purchased.

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Cercospora

Hyphæ and conidia pale brown. The latter tapering towards the apex, usually elongate or acicular, multiseptate.

Leaf Spot of Mangel

Cercospora beticola Sacc.

This disease first appears as small, brown spots, with red margins, irregularly scattered over the leaf lamina. The brown colour of the spots soon changes to grey and afterwards to black. The spots dry up and at the same time cracks and holes appear in the lamina. Finally, the whole leaf becomes dry and curled and falls from the plant. The outer leaves are the first to be affected and then the inner ones. In the meantime, the plant seeks to make good the loss of the old withered leaves by forming new ones, and in this way the head of the mangel becomes elongated and pointed, whilst in the lower part there is little further growth. The disease can also attack the flowering organs and the seeds.

An examination of the spots, when they are of a grey colour, leads to the discovery of tufts of hyphæ which belong to the fungus *Cercospora beticola*. From the tips of these hyphæ, long, multiseptate conidia are abstricted which germinate easily and spread the disease. In the open the conidia die within four months; when dried they can remain viable for eight months. The mycelium also produces sclerotia which are partially embedded in the leaf tissue; in this way the fungus remains alive through the winter to the next growing period.

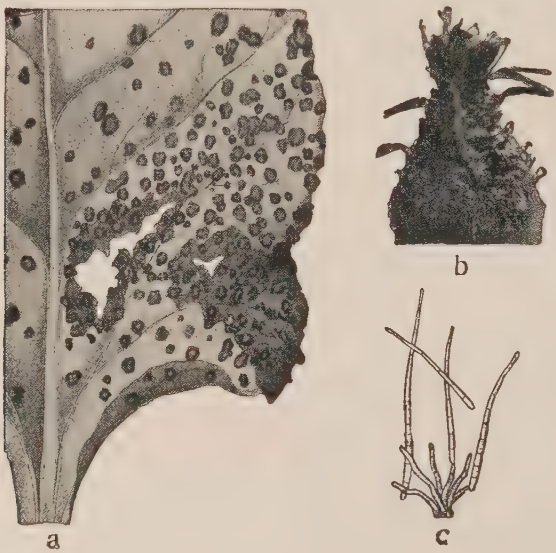

Fig. 369.—Leaf Spot of Mangel.
a, diseased portion of leaf; *b*, pointed head of diseased mangel; *c*, conidiophores abstricting conidia. (*a*, after O. Kirchner and H. Boltshauser; *c*, after B. M. Duggar.)

The disease attacks sugar beet, mangels, and beetroot. It has been known since 1886 in Austria and is now found in many European countries as well as in North America. In 1915 the sugar-beet crop in France was severely attacked and a great reduction caused in the yields.

Control Measures:

(1) The patches of diseased plants in the field should be at once sprayed with 1 per cent. Bordeaux mixture. (2) Seed from infected plants must not be used.

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Leaf spot diseases, similar to the foregoing, are caused by **Cercospora Apii** Fres. on celery, parsnip, and related plants (Halsted, 1896; Prillieux, 1897; Duggar, 1897) and also by **C. Medicaginis** E. & E. on *Medicago maculata* (Hopkins, 1921). **Dendryphium penicellatum** Fres. produces dry, dark brown to black spots on the leaves and stems of the poppy; from these spots, black hyphal tufts arise. The hyphæ are branched and produce three- or four-celled conidia which have sometimes a median constriction.

Leaf Spot of Potato

Cercospora concors (Casp.) Sacc.

This fungus gives rise in summer (from the middle of July) to irregular spots of varying sizes on the leaves of the potato. The spots on the upper side are yellow at first and not sharply defined; on the under surface they are generally covered with a delicate, greyish-violet

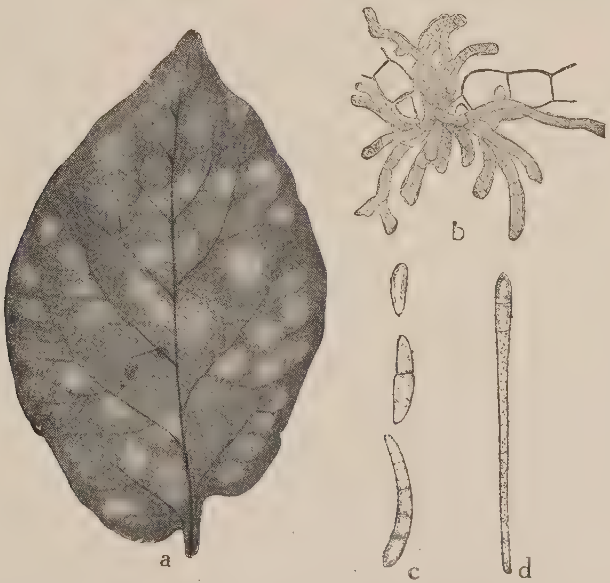

Fig. 370.—Leaf Spot of Potato.
a, diseased leaf seen from above; *b*, cross-section of tuft of hyphæ from the under side of the leaf; *c*, short, and *d*, long conidia. (After P. Lagerheim and P. Wagner.)

felt of hyphæ from which conidia are abstricted. The leaf changes further to a yellow colour and the spots become brownish-black. On the under surface, the conidia are short and blunt, whilst those on the upper surface are fewer in number, longer, and drawn out at one end. As the disease begins early in summer it is able to reduce the crop very considerably. From time to time since 1854 the disease has broken out in Germany, Austria, and France; it has occasionally been observed in Sweden, Denmark, and Finland.

Control Measures:

(1) The haulms from an infected potato field should be removed and burnt. (2) On fields where the disease has appeared, potatoes should not be grown for three years.

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Early Blight of Potato

Sporidesmium Solani varians Vanh.

Few or many, sharply limited, brownish-black spots, which dry up and are often epicentrically zoned, are sometimes found in summer on the leaves of the potato plant. The spots generally appear first on the terminal leaves, later also on the lateral ones. In severe cases the leaves become withered and black and the plant dies prematurely. In certain places the disease has caused great damage, not a single plant or even a single leaf being free of the attack of the fungus.

On the older spots, small tufts of brown hyphæ appear as offshoots of the mycelium, living in the leaves and leaf hairs. The spores are either dark, minute, one-celled, or relatively large, multicellular, and of a very characteristic, narrow, pear-shaped form. Sometimes pycnidia appear later in the autumn; they are embedded in the tissue and contain pycnoconidia which are one-celled, colourless, and of varying shapes and sizes. The shape of the conidia may be ellipsoidal, cylindrical, ovate, pyriform, straight, or slightly curved. The pycnidia enable the fungus to persist over the winter.

The disease occurs mainly on early potatoes with thin leaves and to a less extent on late sorts with thick leaves. It has been known since the latter part of the previous century and was formerly confused with

Late Blight (*Phytophthora infestans*). The disease is especially feared in North America, where it is termed Early Blight (*Macrosporium Solani* Ell. & Mart.). It is also found frequently in Austria, Hungary, Czechoslovakia, Germany, and other European countries, where it is regarded as a not unimportant pest. In Germany the fungus has been named *Alternaria Solani* Sor.

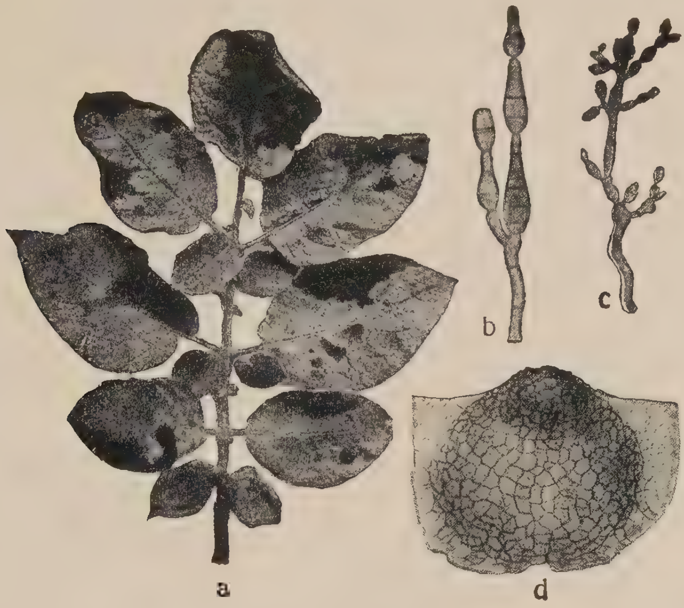

FIG. 371.—*Sporidesmium Solani varians.*
a, diseased leaf of potato; *b*, hyphæ abstricting multicellular conidia; *c*, hyphæ abstricting unicellular spores; *d*, pycnidia embedded in tissue of leaf. (After J. J. Vanha.)

Control Measures :

(1) Collect and destroy the infected potato haulms. (2) Spray with 1 per cent. Bordeaux mixture as soon as the dry spots begin to show (July or August); and if necessary spray again after 2–3 weeks. (3) Tubers from an infected field should not be used as seed.

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In this connection the following fungi may be mentioned: **Macrosporium sarcinæforme** Cav. appears on *Trifolium pratense* and many wild species of clover. Dark brown spots which spread until the whole leaf is affected and finally withers. The disease has been observed in Italy and Germany (Malhoff, 1902). **Alternaria tenuis** Nees. attacks young tobacco seedlings, which become brown, flaccid, and covered with a felt of dirty green hyphæ which finally becomes black and of a velvety appearance. Healthy, adult plants are immune. Abnormally high degrees of moisture in the air and soil favour the destruction of the seedlings (Behrens, 1892).

"Girdle Scab" of Mangel

Oospora cretacea Krüg et al.

This disease is first apparent about the time when the mangel crop is ready to be lifted. Smaller or larger portions of the outside of the mangel are seen to be of a dark colour and covered with a scurfy layer which bears numerous transverse cracks. The mangel is often constricted, usually in the middle, or there may be irregular depressions of varying shapes. The disease is caused by several species of the genus *Oospora* Wallr., especially *O. cretacea* Krüg., *O. rosella* Krüg., *O. intermedia* Krüg., *O. tenax* Krüg., *O. nigrificans* Krüg., and *O. violacea* Gasp. Certain worms (*Euchytræideæ*) often assist in producing the disease, as by feeding upon the root they cause cavities to be formed and so provide

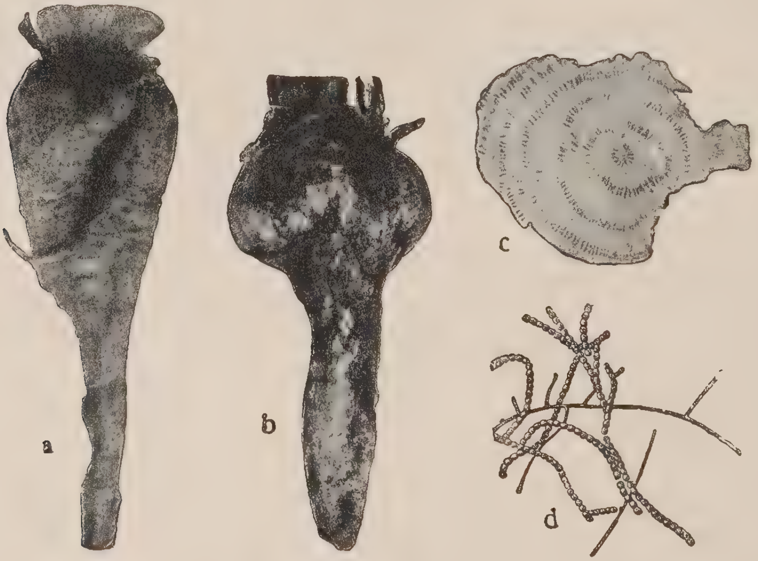

Fig. 372.—*Oospora cretacea.*
a, early, and *b*, later stage of disease; *c*, cross-section of diseased root; *d*, vegetative hyphæ and chains of spores of fungus. (After F. Krüger.)

a point of entrance for the fungi; the fungi themselves are not able to enter the uninjured surface. The disease is most severe after winters with small rainfall and but little cold weather; also a very wet and cold spring and a very dry and hot summer favour the disease. Manuring with nitrate of soda is held to increase the liability to disease, whereas sulphate of ammonia is recommended.

The disease has been observed in a number of places in Germany since the middle of the nineties of last century; in some districts, as between Aschersleben and Hildesheim in 1899 and 1908, the outbreaks have been severe.

Control Measures:

(1) Care should be taken not to infect a healthy field with soil from a diseased one. (2) Wet soils should be drained. (3) The soil should be limed.

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In North America a similar disease, "Beet Scab," is known and the causal organism is said to be *Oospora Scabies* Thaxt. The disease first appears in the form of small, separate or grouped, raised patches on the surface of the beet. The patches extend and to some extent coalesce, giving rise to larger or smaller growths covered with warts. The disease is considered to be identical with a scab of potatoes caused by a fungus of the same name (Sturgis, 1896).

Potato Scab

Actinomyces æruginosus Wr. et al.

This disease attacks growing potatoes, not those that have been lifted and stored; it is sometimes known as "Common Scab." The disease manifests itself in the form of circular or oval patches, or scabs, which are usually sunken and covered by portions of the ruptured skin. The scabs have often an irregular, star-shaped outline and may bear a greyish-white felt which is composed of much-branched hyphæ and chains of spherical or oval conidia. This down-like felt does not absorb water and in a dry atmosphere it shrivels to an impalpable dust. In damp surroundings it develops more strongly and assumes a subdued shade of colour (bluish-green, yellowish-white, bluish-grey, etc.). This form of scab is distinguished from other scab diseases of the potato by the peculiar, unpleasant, earthy smell which is noticeable when the potatoes are being lifted. The disease does not cause the tubers to rot but only attacks the surface, and does not seriously affect the market value.

A number of species of the genus *Actinomyces* (in which genus are also found species which give rise to patches and warts in the tissues of human and animal skin) are stated to be responsible for scab, and each species produces its own type of the disease : (1) *Actinomyces œrugineus* Wr. (" bulging scab ") ; (2) *A. tricolor* Wr. and *A. intermedius* (Krüg.) Wr. (" shallow scab ") ; (3) *A. incanescens* Wr. (" deep scab ") ; and (4) *A. xanthostroma* Wr. and *A. albus* var. *ochroleucus* (Renk.) Wr. (" variable scab ").

The Actinomyces fungi which cause scab are very sensitive to acid conditions so that a sour soil limits the disease, whereas an alkaline one favours it. The application of sulphate of ammonia or other acid-producing manures is therefore to be recommended.

The form of this disease which appears in America is ascribed to the fungus *Actinomyces scabies* (Thaxt.) Güss.

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In North America a disease of maize (Black-Bundle Disease) which at times causes much loss is considered to be produced by a fungus—*Cephalosporium acrenomium* Corda—which is related to the one just described (Reddy and Holbert, 1924).

Spondylocladium atrovirens Harz. (" Silver Scurf ") appears on the surface of the potato in the form of discoloured, silvery, or somewhat darker, patches which often bear numerous small black dots on which conidiophores and conidia develop. The conidiophores produce several whorls of conidia which are greyish-black, obclavate, and have mostly 6–8 septa. Black sclerotia, which were previously given the name *Phellomyces sclerotiophorus* Fr., are developed at a later stage. The diseased patches shrivel and break open. In the case of red-skinned potatoes the colour is often completely destroyed. The mycelium spreads also to the underlying, subepidermal layer of cells. When the moisture and temperature conditions are favourable, the disease spreads rapidly amongst stored potatoes, giving them the appearance of having been dusted with soot. The disease is carried from place to place and from year to year by infected tubers; it was first observed in 1871 in Austria, and is now known in most European countries as well as in North America. In the last-mentioned country it is the cause of very heavy losses, particularly in the eastern States (Appel and Laubert, 1907; Melhus, 1913; Schultz, 1916; Crépin, 1923).

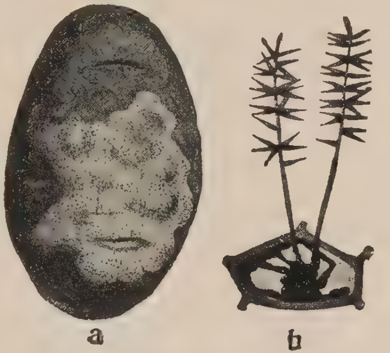

Fig. 373.—*Spondylocladium atrovirens*. *a*, diseased potato tuber; *b*, cell of skin of potato with sclerotium from which conidiophores have grown out. (After O. Appel and R. Laubert.)

Cercospora circumscissa Sacc. produces spots on the leaves, shoots, and fruits of the almond tree (*Amygdalis communis*). The leaf-spots are circular, yellow to brown, sharply defined, with tufts of olive-green conidiophores in the centre. The diseased tissue withers and falls out, so that in severe cases the leaves have a "shot hole" appearance. The spots on the bark of the twigs are roundish in shape; in the case of the fruit the husk only is affected, not the kernel. The conidia are elongate, two- to five-celled, and in a damp atmosphere they germinate in 2–3 hours. The fungus also attacks neighbouring peach and plum trees. The disease is found chiefly in North America and Australia, and it has also been observed in Europe, *e.g.* France and Italy (Pierce, 1892).

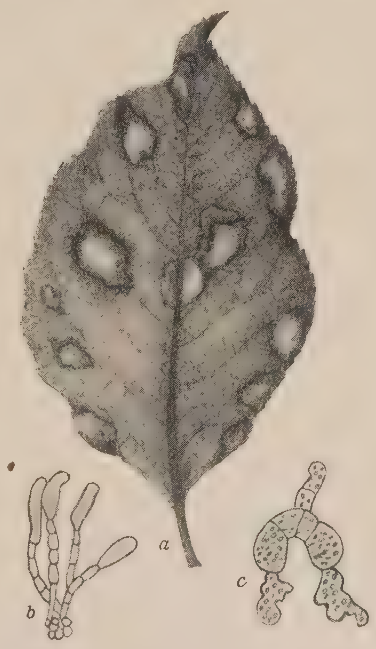

Fig. 374.—*Cercosporella Euonymi*. *a*, diseased leaf; *b*, conidiophores; *c*, conidium, germinated. (Orig.)

Isariopsis griseola Sacc. gives rise to brownish-grey spots, which are generally limited by the veins, on the leaves of the bean. On the under side small brown tufts of conidiophores appear, the latter have enlarged ends and abstrict two- to three-celled conidia.

Cercosporella Euonymi Eriks. produces irregular, translucent spots with brown margins on the upper leaf surface of *Euonymus europæus*. On the under side of the spots, small tufts of branched conidiophores arise. The conidia are bent, almost to a half-moon shape, three- to four-celled; each cell puts out a short, branched germ-tube. The leaves wither prematurely and fall. In 1883 this fungus caused much damage in a nursery near to Stockholm (Eriksson, 1891).

Macrosporium Tomato Cke.—About the time the tomatoes ripen, it is noticed that after continued rain or heavy mists, circular, brown, somewhat depressed spots appear on the fruits. The spots enlarge until the greater part of the surface is affected. In the underlying tissues a weft of mycelium, which is at first colourless and later brown, arises, and from it are produced olive-brown, fertile conidiophores which ultimately cover the spot with a black, velvety down. The spores are divided by longitudinal and transverse septa so that a large number—fifteen or more—of almost cubical cells are formed. The affected fruit is inedible and does not ripen. By means of artificial infection, the fungus has been transmitted to the leaves of a number of sorts of tomato.

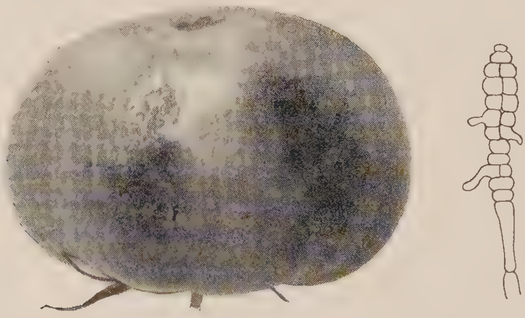

Fig. 375.—*Macrosporium Tomato.* (Orig.)

If the unripe fruit from a diseased plant is brought indoors to ripen or is packed and despatched for sale, the disease may break out in the course of a few days and so be carried from place to place. As soon as the fruits are fully ripe they become immune and cannot spread the disease.

This fungus was first described from America in the eighties of the last century under the name of *Alternaria Solani* (E. & M.) Jones and Grout, whilst in England it was named *Macrosporium Tomato* Cke.; later it was reported from a number of the countries of the Old and New Worlds. In the years 1913–19 it caused much loss in the United States (Florida, New Jersey, Texas, Louisiana), as much as 20–50 per cent. of the crop being unusable (Cooke, 1906; Massee, 1910; Edgerton and Moreland, 1913; Rosenbaum, 1920; Douglas, 1922).

Control Measures :

(1) Use seed from healthy plants. (2) Spray with Bordeaux mixture. (3) Maintain good ventilation in the glasshouses.

A closely related disease, **Macrosporium Foot Rot,** which is ascribed to the fungus *Macrosporium Solani* E. & M., was found in America (Delaware) in 1919 (Rosenbaum, 1920).

Alternaria Grossulariæ Jacz.—This fungus produces small, punctiform spots on unripe gooseberries in May or June. The spots gradually enlarge, until patches of a size of 2 cm. are formed. These patches are of irregular shape and sometimes cover more than half the surface of the berry. Under the skin of the patches there is found a dark, luxuriant,

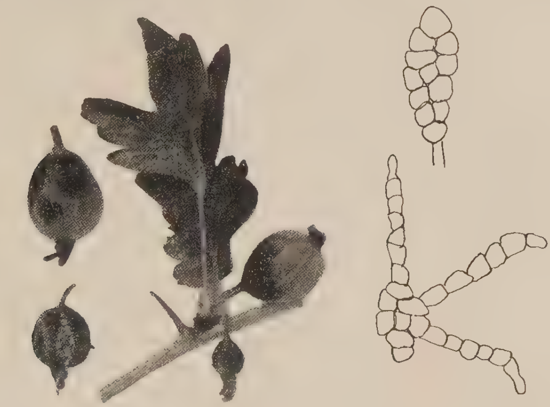

Fig. 376.—*Alternaria Grossulariæ.* (Orig.)

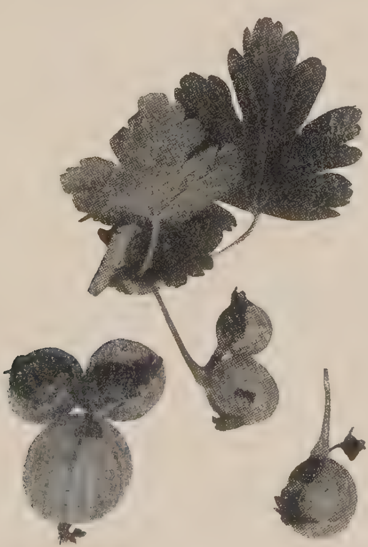

Fig. 377.—*Alternaria Grossulariæ.* Twin and triplet berries. (Orig.)

branching mycelium which does not, however, penetrate deeply into the fruit. Cracks develop in the diseased berries from the middle of June onwards and in these cracks numerous tufts of conidiophores and conidia arise. The conidia are many-celled and germinate easily. The infected berries soon drop. By artificial inoculation with pure cultures of the conidia, it has been possible to produce the characteristic spots on the berries at the end of four weeks. The fungus also gives rise to brown spots on the leaves, and from these spots tufts of conidiophores grow out when the leaves are kept moist under a bell-jar. Leaves on which these lesions appear often fall prematurely in large numbers.

There are considerable differences in the susceptibility of various sorts of gooseberries. Thus in Switzerland it was found that Rote Preisbeere (Roaring Lion), Smuggler and Samson were severely attacked by the fungus, whilst Peace Maker only suffered slightly. Most of the local sorts, about two dozen, were completely resistant.

The fungus was first described from Finland in 1906 (v. Jaczewski) and about the same time it was observed near Hamburg (Brick). In 1909 it appeared in two places in Sweden and in the following year in Switzerland (Schneider-Orelli, 1912).

In certain cases twin- or triplet-berries are produced on gooseberry bushes suffering from this disease.

Control Measures :

(1) Collect and burn all diseased fruits and leaves. (2) Spray the bushes at an early date with Bordeaux mixture.

Numerous other species of the genus **Alternaria** often appear as more or less destructive parasites. **A. Violæ** Sacc. produces greenish or yellowish spots, of varying size, on the leaves of cultivated violets (Marie

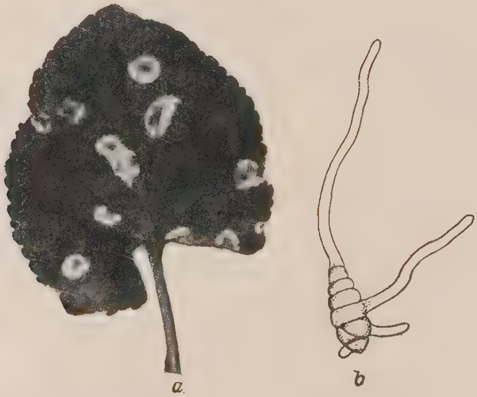

Fig. 378.—*Alternaria Violæ.*
a, diseased leaf ; *b*, germinated spore. (After P. H. Dorsett.)

Louise, etc.). The centre of the spot is covered with tufts of conidiophores. The conidia are six- to ten-celled and germinate easily. In greenhouses in North America and Italy the disease has been very destructive (Dorsett, 1900; Montemartini, 1904). **A. Dianthi** St. & Hall. on *Dianthus caryophyllus:* small, black spots on the leaves and stems; has been observed in North America, Hungary, and Italy.

Elm Branch Disease

Exosporium Ulmi Eriks.

This disease occurs on young trees of different kinds of elm such as *Ulmus montana*, *U. m. exoniensis*, *U. campestris*, and *U. effusa*. The tips of the twigs, or whole twigs, of the infected plants are killed. On the dead parts of the bark are found scattered, black, pustular spore-masses about the size of a pin's head or somewhat larger. These pustules are very numerous in the angles of the twigs. Here and there the bark is torn and bears thin, expanded, ragged crusts.

When a pustule is cut across, a raised, semi-globular stroma is found, from which long hyphæ arise radially. From the tips of the hyphæ conidia are abstricted. The conidia are olive-grey, rounded at both ends, multicellular, with transverse and longitudinal septa; they germinate easily.

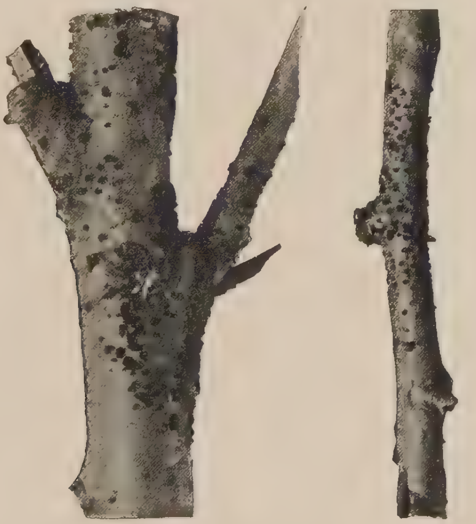

Fig. 379.—Elm Branch Disease. (Orig.)

By means of inoculation tests and out-of-door observations it has been found that the fungus enters the host plant through the young, green, one-year-old shoots. The conidia, which ripen at the same time and germinate readily, adhere in large numbers to the rough surface of the shoot and penetrate into the host plant. After inoculation the fungus lives hidden within the shoot until the following spring and then makes its appearance in April or May in the form of open conidial pustules; by this time the young twig is completely or partially killed. The dead twigs do not fall to the ground but remain on the tree for at least a year. In the second spring, red, *Nectria*-like warts are found on the dead portions of the twigs which had been killed a year previously. It has not yet been decided whether this form in which warts occur is a further development of the conidial stage of the fungus.

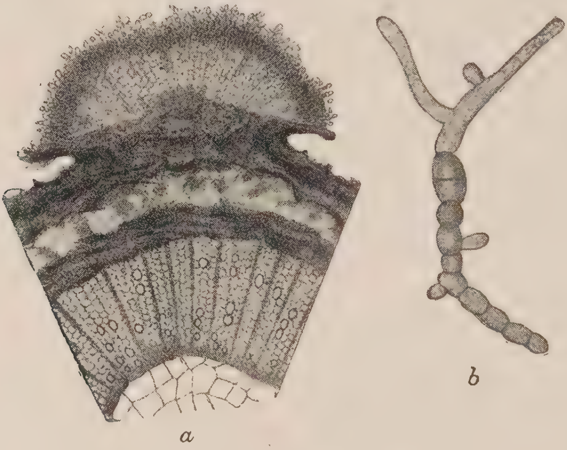

Fig. 380.—Elm Branch Disease.
a, cross-section through a pustule; *b*, germinated spore. (Orig.)

This disease has been known since 1888 in a number of commercial gardens in Sweden and was first described in 1912. In the years 1905–11 it caused serious damage in different Swedish nurseries and new elm plantations.

Control Measures:

(1) Ascertain that the disease does not occur in the nursery from which the young trees are obtained. (2) If the disease is known to be present the trees should be carefully examined in March or April before the new shoots begin to grow, and any dead or diseased twigs should be cut out and burnt. (3) The examination and eradication should be repeated once or twice at intervals of 1–2 weeks.

REFERENCE

Eriksson, J. Über Exosporium Ulmi u. sp. als Erreger von Zweigbrand an jungen Ulmenpflanzen. Myc. Zentr.-Bl., Bd. 1, 1912, 35–42.

Exosporium juniperinum (Ell.) Jacz. on *Juniperus communis*. The terminal twigs are bent ; the needles turn brown and fall readily ; black to olive, velvety cushions of conidiophores occur on the upper, concave side of the brown needles. The conidia elongate and show 3–6 transverse septa ; in 2–3 years the bush dies. The disease has been known in America (Iowa) since 1882, in Finland since 1888, and in Russia since 1896 (v. Jaczewski, 1901).

Chætostroma Cerasi Jacz. occurs on the cherry. On the fruit, near to the stalk, a brown, depressed, shrunken patch appears. On the upper side of the patch are delicate, black fructifications ; the whole of the cherry rots and falls to the ground. This disease appears to be very common in Russia (v. Diakonoff, 1910).

Rhacodium cellare Pers. causes a disease, known in Italy as "Nerume," on edible chestnuts. The tissues of the cotyledons are invaded by a sterile mycelium and become of a black colour. The disease was reported from Italy in 1905 (Peglion).

CHAPTER XIX

FUSARIUM

From the commencement, the stroma lies free on the surface of the host plant and is usually of a waxy or gelatinous nature. The conidia are fusiform, slightly bent, multiseptate. The gelatinous or cottony mycelial covering is colourless at first, later becoming yellow, orange, or brick-red.

REFERENCES

Appel, O., and Wollenweber, H. W. Grundlagen einer Monographie der Gattung Fusarium (Link.) Arb. Kais. Biol. Anst., Land- u. Forstw., Bd. 8, 1913, 1–207.

Wollenweber, H. W. Studies on the Fusarium Problem. Phytop., 1913, 24–50.

—— Die Gattung Fusarium. Handb. Pfl.-Krankh. von P. Sorauer. 4. Aufl., Bd. 3, 164–185.

Fuchs, J. Über die Beziehungen von Fusarium zu anderen Fruchtformen. Zeitschr. Pfl.-Krankh., 1924, 193–220.

Fusarium Diseases of Cereals and Grasses

Fusarium avenaceum (Fr.) Sacc. et al.

In addition to the three species of *Fusarium* found on cereals: (1) *Fusarium nivale* already described under the name *Calonectria graminicola* ("Snow Mould"), (2) *F. rubiginosum* which alone or together with *F. nivale* causes Snow Mould, and (3) *F. rostratum* already described under the name *Gibberella saubinetii*, there are a number of other species, at least twenty, which appear parasitically on the roots, stems, ears, and grains of cereals and grasses. Some of the more important of these species will now be considered.

Fusarium avenaceum (Fr.) Sacc. gives rise, in damp weather in autumn, to salmon or brick-red, fleshy or gelatinous mycelial growths on the ears and panicles of a number of cereals and grasses. Similar lesions are also found on corn stubbles, and occasionally on the seedlings of cereals. The red growth consists of a thick, felted layer of hyphæ from which numerous, erect branches arise. From the tip of each of these branches one or more conidia are formed. These conidia are elongated, three- to five-septate, slightly bent, each end being pointed. Often round, thick-walled chlamydospores are produced from the individual segments of the conidia and serve for the over-wintering of the fungus.

Most of the *Fusarium* diseases which attack the *Gramineæ* such as *Fusarium avenaceum* (Fr.) Sacc., *F. culmorum* (Sm.) Sacc., *F. graminum* Corda, etc., occur mainly on the surface of the parts affected. There are, however, species that penetrate the water-transporting tissues and cause a true wilt. By artificial inoculation several forms have been transmitted to other plants, such as potatoes, tomatoes, beans, etc.

In certain cases these *Fusarium* diseases can give rise to extensive damage to cereal crops. It has been observed that barley and rye plants, sometimes also oats, cease to grow shortly after they have germinated, the colour becomes a pale, greenish-yellow, and the plants in many cases die (root rot of cereals). In other cases the haulms of rye, wheat, and barley bend over and break (foot rot), just as occurs in the attacks of the fungi belonging to the genera *Leptosphæria* and *Ophiobolus*, a description of which has already been given.

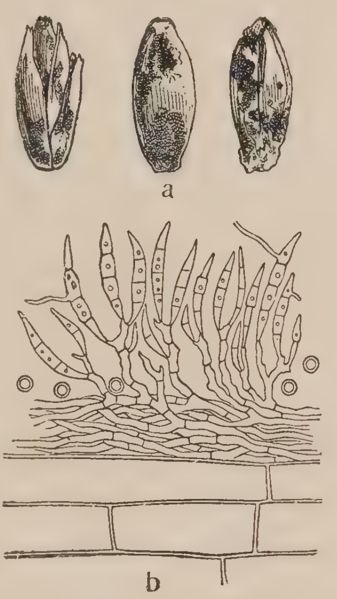

FIG. 381.—*Fusarium avenaceum* on Barley.
a, spikelet and grains with coating of fungus; *b*, portion of the coating. (After M. G. Smith.)

REFERENCE

ATANASOFF, D. Fusarium-Blight (Scab) of Wheat and other Cereals. Journ. Agric. Res., Vol. 20, 1920, 1–32.

Fusarium Maydiperdum Bub.—This fungus is reported from Bulgaria as the cause of a rot in the maize cob. The central axis of the cob withers and the production of grain is checked or may cease entirely. The dead parts of the plant are covered with a delicate network of yellow or rose-coloured hyphæ (Bubák and Kosaroff, 1911). Several other diseases of this nature occurring on maize have been reported from North America.

Fusarium Diseases of Legumes

Fusarium vasinfectum Atk. et al.

This disease has lately been observed in several places on peas, and is noticeable because of the sudden and rapid wilting of the plants; it may appear as early as May.

Some of the young leaves, and soon afterwards the stems, become pale and flaccid, and in the end the whole plant wilts and falls to the ground. The condition is found mainly towards the end of June, which has led to the name "Midsummer Disease" being given to it in Holland. The disease starts at the neck of the root in the form of one or several dark-coloured, disintegrating streaks, which often extend above the soil-level. The fungus enters by existing wounds or cracks, or at the points where lateral roots arise, and spreads to the fibrovascular bundles and finally through the whole plant. The vessels become blocked by a yellowish, gummy substance. Such *Fusarium* diseases, which follow the water-transport system of the plant, are also named "Tracheomycoses."

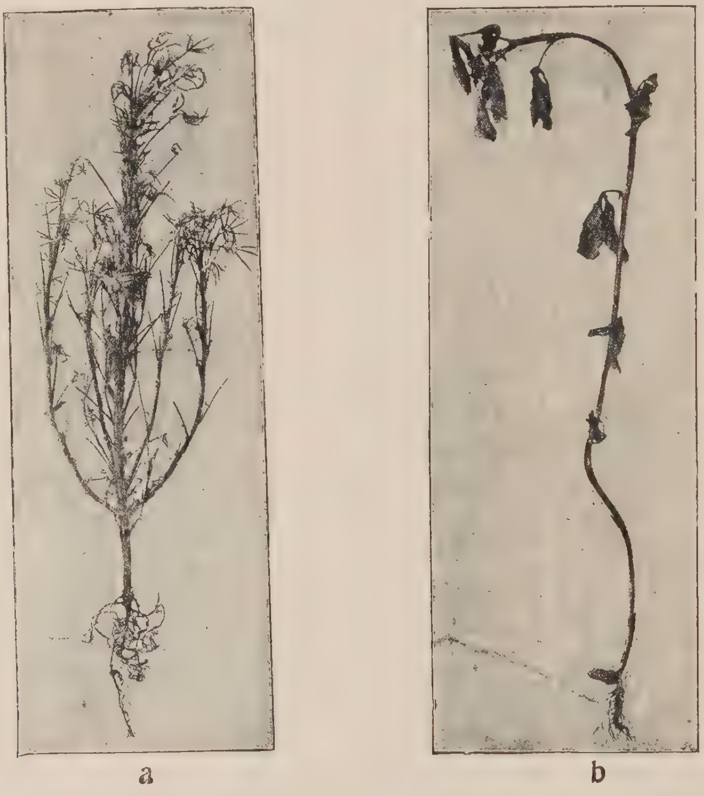

Fig. 382.—Fusarium Disease of Legumes.
a, diseased lupine plant; *b*, young, artificially infected field-bean plant. (After G. Schikorra.)

The fungus spreads to the adjoining cells and produces a considerable number of large, thick-walled resting-spores (chlamydospores) by means of which the fungus remains alive during the winter. When the tissue that has been permeated by the mycelium is exposed, there grows from it an aerial mycelium on which small (one- to four-celled) and larger (three- to six-celled) conidia are developed. By means of these conidia, the fungus spreads from plant to plant during the growing season.

Three different species of *Fusarium* are stated to be the cause of the disease: *F. vasinfectum* Atk., *F. redolens* Woll., and *F. falcatum* App. & Woll.

Similar diseases, usually termed " wilts," occur on a large number of other kinds of legumes; for example, on species of *Faba*, *Vicia*, *Lupinus*, *Trifolium*, and *Lathyrus*. In the case of horse-beans, there is a wilting and collapse of the plants if they become infected at an early stage. If the plants are, however, older the parasite is not able to bring about the fall of the stem and only the leaves wilt. In the case of lupines, such as *L. angustifolius*, *L. perennis* and *L. mutabilis* the disease appears on young plants shortly before flowering, and also on older plants that have already produced pods. The stronger stems remain, as a rule, erect. Various species of *Fusarium*, such as *F. euoxysporum* Woll., *T. tracheiphilum* (Erw. Sm.) Woll. are given as the cause of the disease.

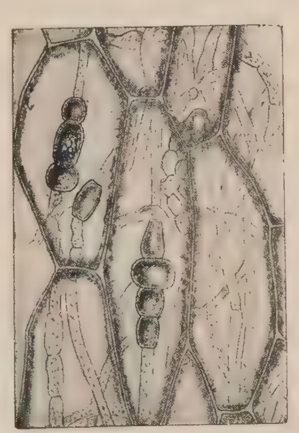

FIG. 383.—Cells from stem of Pea Plant suffering from Fusarium Disease, showing Mycelium and Chlamydospores. (After G. Schikorra.)

It has been proved experimentally that a *Fusarium* found on one species of *Leguminosæ* can be transferred to other species.

Control Measures:

(1) Seed of low germinating power should not be sown. (2) If patches of disease occur in a field, the diseased plants should be removed and destroyed. (3) All parts of diseased plants remaining after harvest should be collected and burnt. (4) Leguminous crops should not be grown for two or three years on soil known to be infected.

REFERENCE

SCHIKORRA, G. Die St. Johanniskrankheit der Erbsen. Arb. Kais. Biol. Anst., Land- u. Forstw., Bd. 5, 1907, 157–173.

Dry Rot of Potato

Fusarium cœruleum (Lib.) Sacc.

This disease often appears during the winter on stored potatoes in the form of small, raised or sunken spots on the skin. The spots are covered with white, bluish, or pale red masses of hyphæ which produce conidia. The conidia are falcate, pointed at both ends, and multiseptate. They germinate easily, within a few hours, under favourable conditions, and spread the disease to surrounding plants. In addition to these rapidly-germinating conidia, other more permanent spores—chlamydospores—gradually develop in the pustules. These chlamydospores are formed from certain of the mycelial hyphæ and are intercalated, often several together in a row; they only germinate after a resting period. The inside of badly-attacked tubers is changed within a short time into a soft, rotten mass. As a rule, the disease is not noticeable at harvest time but remains dormant and appears later.

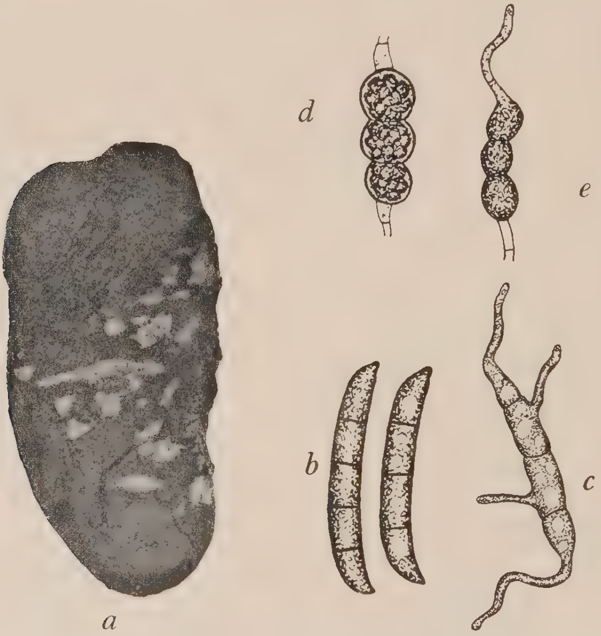

Fig. 384.—Dry Rot of Potato.

a, tuber covered with spots of fungus; *b*, conidia; *c*, germinating conidia; *d*, chlamydospore; *e*, germinating chlamydospore. (*a*, after Board of Agriculture; *b*–*e*, after G. H. Pethybridge.)

It was formerly thought that this fungus was the conidial stage of the ascomycete fungus *Nectria Solani* R. & B., which had, in addition, conidial forms belonging to the genera *Monosporium* and *Cephalosporium* (Reinke and Berthold, 1879). Later investigations have, however, shown that this view is not correct.

In America, several other species of *Fusarium* are said to cause the disease: *Fusarium oxysporum* Schl., *F. trichothecioides* Wr., *F. eumartii* Carp., and *F. radicicola* Wr. (Carpenter, 1915; Pratt, 1916).

This disease possibly appeared to a serious extent in Germany and France in 1907. Large consignments of seed potatoes, sent in 1908 from these countries to the Transvaal, were so badly diseased that the Transvaal Government found itself obliged, in the autumn of the same year, to order that any consignments of potatoes that were infected by this disease, to an extent of 1 per cent. or more, must be destroyed or returned to the exporters.

Control Measures :

(1) Potatoes should be allowed to dry well before being stored ; a close, damp atmosphere should be avoided in the storage place. (2) The stored potatoes should be examined from time to time and all diseased tubers removed. (3) All diseased potatoes, even those only slightly affected, must be well boiled before being fed to animals. (4) Healthy tubers only must be used for seed. (5) A field on which diseased potatoes have been found during one of the last 3–4 years is not fit for another potato crop.

REFERENCES

Appel, O., and Fuchs, J. Zur Kenntnis der Fusariumfäule der Kartoffeln. Mitt. K. Biol. Anst., Land- u. Forstw., 1913, April, 16–17.

Pethybridge, G. H., and Lafferty, H. A. Further observations on the cause of the Common Dry Rot of the Potato Tuber in the British Isles. Sci. Proc., Roy. Dublin Soc., 1917, June, 193–222.

Fusarium diseases are also found on a large number of other horticultural crops : thus beans (Burkholder, 1919, 1924), onions (Prillieux, 1899 ; Clinton, 1906, 1916 ; Walker, 1919, 1924), tomatoes (Edgerton, 1918 ; Pritchard, 1922 ; Shapovalov, 1924), cherries (Aderhold, 1901), raspberries (Osterwalder, 1911), mulberry trees (Briosi and Farneti, 1904), vines (Rives, 1924 ; Foëx, 1926), acacias (Hennings, 1902), carnations (Sturgis, 1898 ; Delacroix, 1900), gladioli (Massey, 1926), etc.

CHAPTER XX

RHIZOCTONIA

THE fungi belonging to this genus produce an expanded, felt-like, violet or brown mycelium on the surface of the roots or other underground parts of the plant. The reproductive organs, as far as they are known, are incompletely developed.

Root Rot of Root Crops

Rhizoctonia violacea Tul.

This disease attacks a number of root crops, especially carrot, sugar beet, mangel, kohl-rabi, and turnip, and first makes its appearance about harvest-time. It is then seen that here and there in the field the leaves have wilted. An examination of the root of the plant shows a violet felt, composed of hyphæ, which is continuous round the upper, middle, or lower part of the root. The parts of the root that are not affected, or only slightly, develop more or less normally and retain their natural colour. On the other hand, the part covered by the felt is often constricted and thinner than the rest. When an infected root is drawn from the ground, the particles of soil adhere to the parts covered by the felt and cannot be removed without detaching some of the felt and adjacent portions of the root tissues.

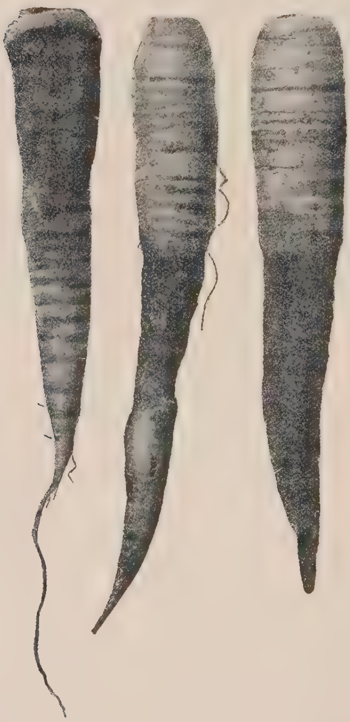

FIG. 385.—Three Carrots attacked by Root Rot, the first on the upper part, the second in the middle, and the third on the lower part. (Orig.)

When the hyphæ have grown older they contain a red colouring matter, but the walls are usually colourless. In the felt lie numerous small, brownish-black, sclerotia-like structures which are composed of short, considerably branched, interwoven hyphæ. These " sclerotia " gradually free themselves from the root and enter the surrounding soil; they undoubtedly play a part in the spread of the disease.

Inoculation experiments, carried out in the vicinity of Stockholm, have shown that there are at least two specialized forms of the fungus: (1) f. sp. *Dauci*, which occurs on *Daucus Carota* (in a varying degree on different varieties of carrot) and on *Beta vulgaris* (and to a lesser degree on lucerne and potatoes). This form also occurs on a number of weeds such as *Erysimum cheiranthoides*, *Stellaria media*, *Urtica dioica*, *Galeopsis Tetrahit*, *Myosotis arvensis*, *Sonchus arvensis*, *S. oleraceus*, and *Chenopodium album*, but not on parsnips or clover; and (2) f. sp. *Betæ*, which occurs on *Beta vulgaris* and, in addition, is transmissible to *Brassica Rapa* and *Daucus Carota* but not to red clover.

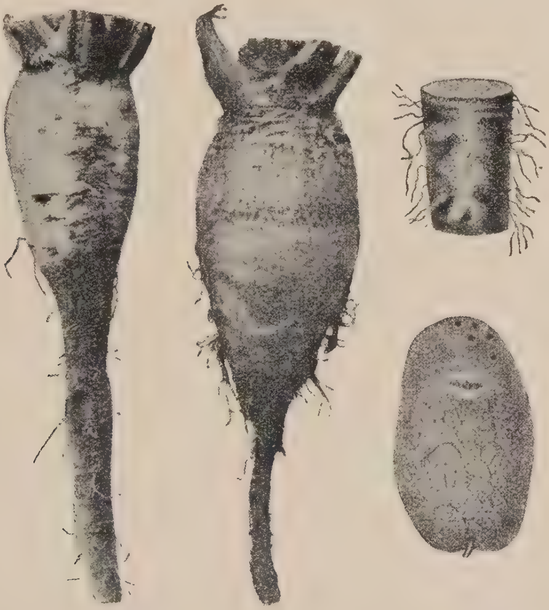

FIG. 386.—Sugar Beet (left), Mangel (middle), Portion of Root of Lucerne (right, above) and Potato (right, below) infected with material from Carrots attacked by Rhizoctonia Fungus. (Orig.)

The specialized form *Dauci* occurs on the root as well as on the lower part of the stem of the above-mentioned weeds. On the roots the fungus shows itself mainly in the form of black dots. At the neck of the root, especially with the first five weeds mentioned, a thick, pale, flesh-coloured tumour arises which completely covers the stem and sometimes spreads on to the surface of the soil. Many of the weeds attacked by the fungus, for example *Sonchus oleraceus*, *Stellaria media*, *Erysimum cheiranthoides*, and *Urtica dioica*, were still fully alive at harvest-time, the stems and leaves being green and in one case (*Erysimum*) with living flowers. The other varieties had, however, wilted.

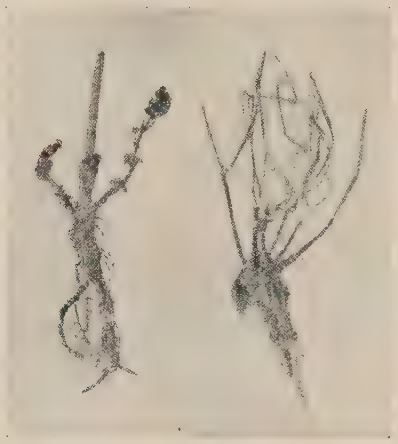

Fig. 387.—Portions of *Urtica urens* (left) and *Stellaria media* (right) attacked by Rhizoctonia Fungus of Carrot. (Orig.)

It was observed, several years later, on diseased portions of the weeds which had been stored in spirit, that mycelial outgrowths arise from the bright red felt on the stems. The resemblance of these outgrowths to basidia and basidiospores was so striking that the suggestion was made that as in the case of *Rhizoctonia Solani*, where there is a *Hypochnus* stage (*H. solani*), so here there is also a *Hypochnus* stage (*H. circinans*). Later experiments carried out in Holland with fresh material of the same kind have led to the above suggestion being questioned, as no basidia, but only hyphæ, were found. Further experiments are required to decide this point.

Control Measures :

(1) When the crop is harvested, all roots that show signs of disease must be taken out and either destroyed or rendered harmless by boiling. (2) In the case of patches of disease in a field, the area should be marked and later disinfected with carbolic acid solution (50 gr. to 10 litres water ; 40 litres of solution to 15 sq. metres of ground). (3) Roots lifted from the vicinity of the infected parts of the field should be examined from time to time during the winter and any that show signs of disease should be removed. (4) A field from which infected roots have been obtained should not be used for any crop that is susceptible to the disease, until at least four years have elapsed. (5) Dung made by animals to which diseased roots have been fed should not be used in a fresh state.

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Prillieux, E. Rhizoctonia violacea (DC.) Tul. Malad. Pl. Agric., T. 2, 1897, 144–157.

Duggar, B. M., and Stewart, F. C. The Sterile Fungus Rhizoctonia. Corn. Un. Agr. Exp. Stat., Bot. Div., Bull. 186, 1900, 51–76.

ERIKSSON, J. Einige Studien über den Wurzeltöter (Rhizoctonia violacea) der Möhre. Centr. Bl. Bakt., Abt. 2, Bd. 10, 1903, 721–738, 766–775.

—— Etudes sur la maladie produite par Rhizoctone violacée. Rev. Gen. Bot., T. 25, 1913, 14–30.

DUGGAR, B. M. Rhizoctonia crocorum (Pers.) DC. and R. Solani Kühn (Corticum vagum B. and C.) with notes on other Species. Ann. Miss. Bot. Gard., Vol. 2, 1913, 403–458.

VAN DER LECK, H. A. A. Contribution à l'étude du Rhizoctonia violacea. Med. R. Hoog. Land. Sch., T. 12, 1917, 94–112.

RICHARDS, B. L. A Dryrot Canker of Sugar Beets. Journ. Agr. Res., Vol. 22, 1921, 47–52.

Root Rot of Asparagus

Rhizoctonia Asparagi Fuck.

This disease shows itself at the beginning of the new season's growth by a general weakness of the plants when they begin to shoot. The

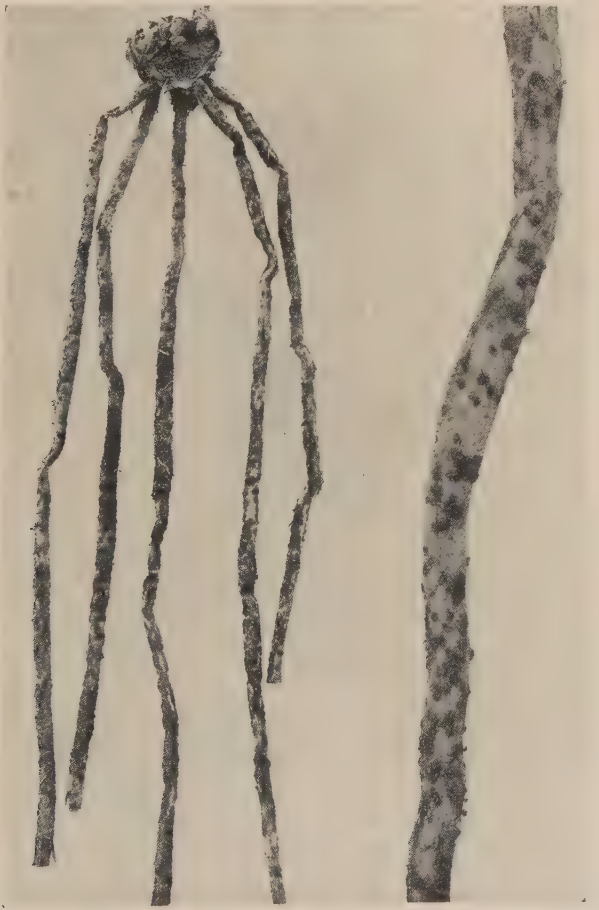

FIG. 388.—Root Rot of Asparagus. (Orig.)

number of young shoots is abnormally small and the stalks that are formed are stunted, dirty yellow, and spotted; the formation of shoots soon ceases entirely. On examination, a violet-coloured felt of varying thickness is found on the surface of the roots. In certain places the hyphæ are closely interwoven, forming dense, hemispherical masses (sclerotia); in France the larger sclerotia are termed "Corps tuberoids," and the smaller ones "Tubercles miliaires." From the sclerotia delicate hyphæ spread on all sides into the soil. When these hyphæ reach a branch of the root that is not already infected they adhere to it and form fresh sclerotia; the hyphæ penetrate by means of their branches into the interior of the roots. The cortex of the root is killed completely, leaving only the central cylinder. In the end, the root has the appearance of a hollow, shrunken tube inside of which is the detached, thread-like central cylinder.

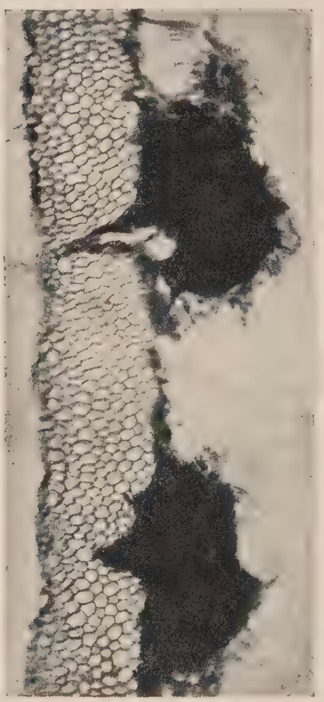

Fig. 389.—Root Rot of Asparagus. Section of the periderm of the root showing two sclerotia. (Orig.)

The underground mycelium seems to be able to ensure the carrying-on of the fungus for an unlimited time. No specific reproductive organs are known, unless the fungus *Diaporthe* (*Leptosphæria*) *Asparagi* Fuck. found on decayed roots of asparagus in Nassau (Hattenheim) many years ago (Fuckel, 1869), but not since met with, is the reproductive stage.

Formerly, it was generally believed that the asparagus *Rhizoctonia* is identical with the forms on *Crocus*, *Medicago*, *Trifolium*, *Beta*, etc. (Duhamel, 1728; Delacroix, 1903). Recent experiments carried out in Sweden in the open do not support this view, as the results obtained when artificial inoculations are carried out under ordinary field conditions do not give a definite proof of the identity of the forms. The asparagus *Rhizoctonia* must therefore be regarded as an independent species.

This disease was first reported from France (du Boudaroy, 1782). In 1869 it was met with in Germany (Fuckel) and at the end of last century in North America, Belgium, and again in France, all about the same time.

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Eriksson, J. Etudes sur la maladie, produite par Rhizoctone violacée. Rev. génér. Bot., T. 25, 1913, 14–30.

—— Fortgesetzte Studien über Rhizoctonia violacea DC. Ark. Bot., K. Vet. Ak., Stockholm, Bd. 14, Nr. 12, 1915, 16–29.

The closely related fungus **Rhizoctonia crocorum** DC., which attacks the bulbs of saffron (*Crocus sativus*) and causes what in France is termed " mort du safran," has a historical interest. The mycelium of the fungus gives rise to white, cottony masses on the inner side of the bulb scales. Branches from the mycelium gradually penetrate to the interior of the bulb scales, the white colour finally changing to violet. In the end, the fungus causes the complete destruction of the bulbs.

The disease, which is frequently discussed in the old French literature, has now lost much of its interest, as the cultivation of saffron has, in consequence of the damage caused by the fungus, been abandoned by many growers since the beginning of the present century (Delacroix, 1903).

CHAPTER XXI

DISEASES NOT FULLY INVESTIGATED

Grey Spot of Oats

It is sometimes seen in a field of oats in early summer that portions of the crop are suffering from a disease which produces the following symptoms. The leaves of the plants are spotted and may wither completely before the panicles have been formed. Later, the plants either die or produce stunted panicles with empty, withered spikelets. The large, brownish-grey spots on the leaves are at first separated from one another, but they increase in size until the greater part, or the whole, of the lamina is dead. As a rule, the spots first appear on the base of the second leaf and then spread gradually to the upper leaves.

Usually, small, black dots composed of hyphæ are found on the grey leaf-spots. These hyphæ emerge from the stomata and consist of erect, dirty brown conidiophores which are branched in a zigzag manner and from which are abstricted one-celled or transversely divided conidia, also of a dirty brown colour.

The disease generally reappears on the same parts of the field when a crop of oats is again grown. Sometimes there are similar appearances of disease on summer wheat when it is grown on such fields. The disease is often found on peaty soils and on soils of an alluvial, sandy nature, near to lakes and running water. Not infrequently, it occurs on sloping ground between elevated and low-lying soils. Low temperatures during the early growing period of the crop are favourable to the disease.

The appearance and intensity of the disease are connected with an excess of lime in the soil solution. By the addition of potassium permanganate, manganese sulphate, ammonium sulphate, and ammonium chloride, as well as by the use of well-rotted dung, the disease can be checked. Nitrates, on the other hand, favour the disease (O. Arrhenius, 1923, 1924). Because of the foregoing, it is sometimes considered that the fungus which is present—*Scolecotrichum graminis*—cannot be regarded as the primary cause of the disease. Different sorts of oats suffer to varying degrees from the disease ; Mesdag, Duppauer, Deutscher Moorhafer, etc., appear to be very resistant.

The disease has been known in Sweden from south to north for a long time, but has not, until recently, attracted attention. It has also been recorded in Denmark, Holland, and Germany, especially in the years 1908 and 1909 in Posen and West Prussia.

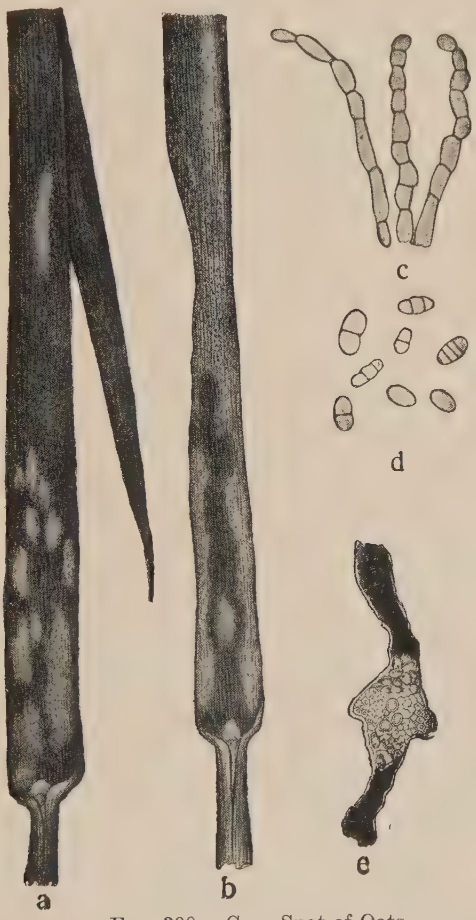

Fig. 390.—Grey Spot of Oats.
a, early, *b*, later stage of disease ; *c*, hyphæ from tuft of conidiophores ; *d*, conidia ; *e*, section of leaf almost killed. (*a–d*, Orig. ; *e*, after F. Krause.)

Control Measures :

(1) On soils where there is a tendency for the disease to occur, resistant sorts of oats should be grown. (2) Fields that are shaded should be avoided. (3) The use of the sulphates and chlorides of manganese and ammonia is the best means of combating the disease ; then

comes well-rotted dung; about ½ cwt. per acre of manganese sulphate should be used. On the other hand, all nitrates, as well as manganese dioxide, serve to further the disease.

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Die-back Disease of Elm

This disease is characterized by the withering and premature fall of the leaves of elms (*Ulmus montana*, *U. campestris*, *U. hollandica*, *U. pitteursii*). These destructive changes sometimes take place very rapidly; in one case near Aachen in 1927 a forty-year-old tree lost its healthy green appearance in the course of three days, and at the end of a week was quite bare of leaves. In other cases the destruction is more gradual and the tree may live for weeks or months.

Scientific investigators are not in agreement as to the primary cause of the disease. On the one hand, it is held that the disease is a true bacteriosis, as it was found on examining branches, stems, and roots that had been sawn across that the annual rings of the previous three or four years were interspersed with dark dots which often formed continuous rings. The exposed wood was, in many cases, traversed by dark brown and reddish streaks. The wood vessels and the cells of the adjoining parenchyma and medullary rays were filled with a dark brown or reddish-yellow material in which, by appropriate methods of staining, coloured cocci and diplococci were distinguished. Sometimes, also, rod-shaped, bacteria-like bodies (possibly genetically related to the cocci) and portions of the mycelium of the fungus *Graphium Ulmi* Schwarz. were found. Pure cultures of the cocci were prepared on a number of different substrata and with them detached branches and growing trees were inoculated. After some time, these inoculations gave rise to the characteristic symptoms of the disease, which was held to indicate the bacterial nature of the outbreaks. The bacterium has been named *Micrococcus Ulmi* Bruss. It is supposed that natural infection takes place generally through the roots and that from this point the bacteria pass in the transpiration stream to the vessels (Brussoff, 1925–1928).

On the other hand, it is held that the disease is not bacterial in nature but is caused by the fungus *Graphium Ulmi* mentioned above. Careful examination of a number of samples of wood taken from diseased elms growing at Breslau, Nuremberg, Berlin, etc., did not lead to the isolation of bacteria, but by appropriate methods it was possible in all cases to cultivate the fungus *Graphium Ulmi* from the samples of wood which showed typical symptoms of disease. The natural mode of infection of the tree is, according to this view, through the leaves, the spores of the fungus sending their germ-tubes through the stomata. Inside the leaf a mycelium develops from which conidiophores arise and pass out through the stomata. After a fortnight, numerous *Graphium* colonies (coremia) are formed (M. Schwarz, 1922; Stapp, 1928; Wollenweber and Stapp, 1928).

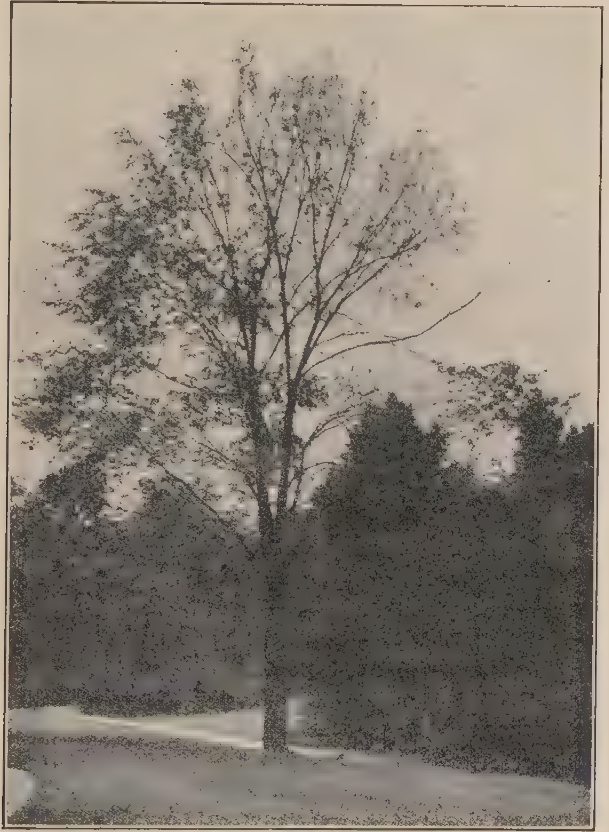

FIG. 391.—*Ulmus montana* (forty-eight years old) in the town park, Steglitz. The right side of the tree already shows distinct symptoms of disease. (After H. W. Wollenweber and C. Stapp.)

In the summer of 1926 a similar disease appeared suddenly in different places in Aachen on silver maple (*Acer dasycarpum*) and lime (*Tilia intermedia* and *T. platyphyllus*) and has been attributed to the same fungus as that which causes the elm disease (Brussoff, 1926).

The final solution of this much-discussed question as to the origin of the disease will probably not be long delayed as much attention is being paid to it in different countries. Possibly two distinct forms of the disease have to be reckoned with, according to the locality : the one form a bacteriosis (*Micrococcus Ulmi*) and the other *Graphium Ulmi*, just as in the case of the apple and pear there is a bacterial canker (*Bacterium Mali* and *Piri*) and a *Nectria* canker (*Nectria galligena*).

This disease was first observed in Holland in 1922 and was then described. From the appearance of the annual rings it was found that the disease had also occurred in the years 1920, 1919, and 1918. About the same time, it appeared in a number of places in Germany such as Westphalia, the Rhine Province, near to Potsdam, Bremen, Nuremberg, Aachen, etc., as well as in Belgium, France, and England (M. Wilson and M. J. F. Wilson, 1917).

Control Measures :

The only possible method of control would appear to be to fell and burn affected trees (Day, 1927). On account of the disease the importation of elms from the continent of Europe has been prohibited since January 15, 1927, in the case of England and Wales, and since February 1 of the same year in the case of Scotland (Statutory Rules and Orders, 1926, No. 1636, 1927, and Statutory Rules and Orders, No. 31, 3, 1927).

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Leaf Roll of Potato

In summer, about the end of July or the beginning of August, it is noticed that some of the plants in a field of potatoes have an abnormal appearance, the leaves being folded or rolled lengthwise in an upward direction so that in some cases the leaflets form a tube. Plants affected in this way may appear singly in the field or in larger or smaller patches. Owing to the paler colour of the under side of the leaves, the diseased plants stand out clearly from the dark green healthy ones and the whole field has a variegated appearance. In some varieties the rolled leaves have a yellowish or reddish tint, especially on the under side of the midrib.

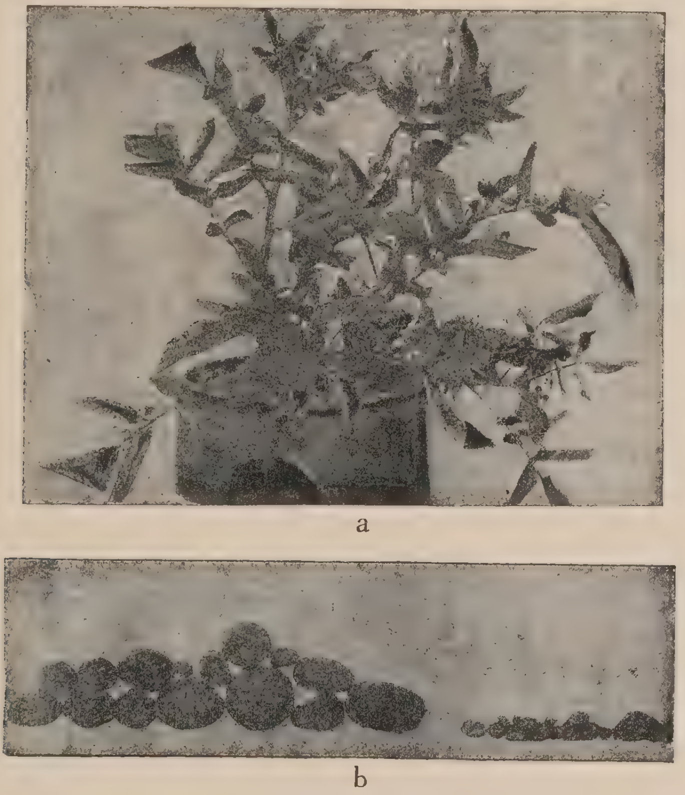

Fig. 392.—Leaf Roll of Potato.
a, diseased plant; *b*, crop from a healthy plant (left) and from a diseased plant (right). (After R. Schander.)

In the first year of the disease the affected plants give, as a rule, an almost normal crop of apparently healthy tubers although the number of potatoes may be small. If, however, the tubers from such a crop are used as seed in the following year, it is found that whilst the eyes produce, mainly, normal underground stems the shoots above ground are stunted and the leaves begin to roll earlier than they did in the first year of the disease. Tubers may be formed, but generally they are small, often not bigger than a hazel nut. Sometimes the underground stems are shortened and the tubers are formed close to the stalk. Not infrequently the eyes of these small tubers produce underground stems on which tubers arise. If the tubers from the second year of disease are used as seed in the following year it is often found that the shoots which are produced develop so badly that they fail to emerge from the soil, or there may be no formation of shoots at all.

The disease was first noticed in Westphalia and the Rhine Province in 1905, and it was then considered to be a consequence of the abnormally dry seasons of 1904 and 1905. Soon afterwards, however, it was observed in other places and later it was reported from most European countries as well as from America.

At the beginning of 1908, the disease was regarded in Germany with alarm, and fears were expressed that disease-free potatoes would not be available for seed. These fears proved, however, to be groundless, for although in many districts where the intensive cultivation of potatoes was practised the outbreaks of disease were serious, there were other districts in which the crops were healthy. It was noticed, nevertheless, that the disease reappeared in places where it had once been severe and that the various sorts of potatoes were, with few exceptions, attacked to the same degree.

When the stem of a diseased potato plant is divided crosswise it is sometimes found that the ring of vascular bundles is coloured yellow, and a thin section of such a stem may show mycelium to be present in the yellow ring. Similarly, the vascular bundles of a diseased tuber are often found to be of a yellow colour, especially towards the end where the tuber is attached to the underground stems. The mycelium has been regarded as belonging to various species of the genera *Fusarium* (Appel, 1906; Spieckermann, 1908; Köck and Kornauth, 1910; Himmelbauer, 1912), *Verticillium* (Appel and Wollenweber, 1909), *Solanella* (Vanha, 1910), etc.

In many cases, however, no mycelium has been found in the vascular bundles nor in the other tissues of the potato, and on this account the parasitic nature of the disease has been questioned and other suggestions offered. Thus the disease has been regarded as due to the use of unripe seed (Hiltner, 1905; Sorauer, 1908; Stoermer, 1910), or to the abnormal enzymatic properties of the cell sap (Grüss, 1907; Sorauer, 1908), or to injury of the roots, resulting in a deficient supply of water, so-called "pathological, adoptive mutation" (Hedlund, 1910). Other suggestions made in this connection are "phlœm necrosis," in which there is a shrivelling and lignification of the phlœm (Quanjer, 1913; Schultz and Folsom, 1921), excessive and unsuitable manuring with potash or other salts (Bibraus, 1910; Hiltner, 1911), and finally, the so-called "degeneration" of the plants owing to continued vegetative propagation (Schultz-Soest, 1905). According to all these hypotheses, the mycelium, if present, must have entered subsequently and cannot be regarded as the real cause of the disease.

Leaf Roll was originally placed amongst the "virus" or "degeneration" diseases of the potato. It was not until 1906 that Leaf Roll was regarded and described as an independent form of disease. Since this time, however, the experience gained in various countries leads to doubt being thrown upon the specific nature of the disease.

Differences in the appearance of the disease, development of the haulm, the rolling of the leaves, the attachment of the tubers (with and without stolons), etc., as well as the differences which occur in certain cases in the economic aspect of the disease, gave rise to the suggestion that other forms of disease of different characteristics are hidden in Leaf Roll. This view has gradually received considerable support. In 1916 a special form of the disease (Verticillium Disease) was reported from Ireland (Pethybridge) and more recently in Holland eight different forms have been described and illustrated—Leaf Roll, Marginal Leaf Roll, Intervenial Mosaic, Aucuba Mosaic, Common Mosaic, Crinkle, Stipple Streak and Leaf Drop Streak (Quanjer, 1924).

Control Measures :

(1) The seed should be obtained from a crop in which the disease has not appeared. (2) A field in which potatoes suffering from this disease have been found should not be planted again with potatoes for the next two or three years. (3) If the disease is noticed in a growing crop the infected plants should be removed and destroyed or else the patches where they occur should be marked and the produce kept separate from the healthy potatoes at harvest-time. (4) Care should be taken that the soil from an infected field is not carried by workers, animals, or implements to any other field where potatoes are to be grown in the following year.

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A disease known in Ireland as "Yellowing" (Yellow Blight) is similar to the above and the cause has not yet been explained. The affected plants begin to yellow about the middle of July and die prematurely, producing either a very small crop or none at all. So far no parasite has been found. Growing the potatoes on high ridges and the provision of good drainage operate against the disease.

For some years past, several potato diseases have been observed but have not yet been thoroughly investigated. One of these, named "Buntwerden," has been known in Germany since 1897, whilst two others, "Internal Disease" (Horne, 1910) and "Sprain or Streak Disease" (Pethybridge, 1912), have been known in England, America, and Australia since about the same time. These diseases all exhibit similar characters. The tubers show no outward signs of injury, but when cut across brown circular patches ("Buntwerden" and "Internal Disease") or narrow streaks ("Sprain") are discovered in the flesh; in the latter disease the streaks are often arched and confluent. Recently the view has been taken that these forms of disease are all caused by the bacterium *Pseudomonas solanioleus* Paine (Paine, 1923). On the other hand, it is maintained that the occurrence of mycelium or bacteria in, or near, the diseased tissues has not been proved. The protoplasm of the cells has nevertheless been observed to be turbid and of a brownish colour, and it has been stated that in some cases round, plasmodia-like bodies have been found in the diseased cells, thus suggesting the presence of one of the lower fungi (*Chytridiaceæ*). Such diseased potatoes are not suitable for human food; if used as seed, then 6–22 per cent. of the plants are similarly affected.

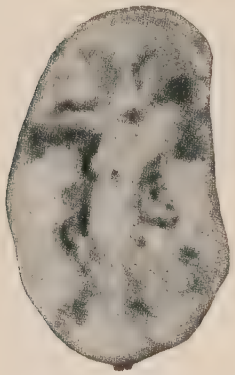

Fig. 393.—Potato attacked by sprain. (After A. S. Horne.)

Mosaic Disease of Tobacco

In summer, 2–3 weeks after planting, some of the tobacco plants acquire a peculiar, abnormal appearance. The leaves show an irregular mottling, some patches being dark green and opaque, others pale and translucent. When held against the light, the diseased leaves have a characteristic "mosaic" appearance. The dark green areas grow vigorously and develop into slightly raised humps, whilst the light areas almost entirely cease to grow. The diseased plants remain very much behind the healthy ones in general growth.

Diseased and healthy plants grow side by side irregularly, yet the disease does not pass, of itself, from one plant to another. If, however, a piece of infected leaf or the sap from such a leaf reaches a wound on the leaf or stem of a healthy plant the latter shows similar symptoms of disease after some 3–6 weeks, according to the age of the healthy plant. The disease does not appear, moreover, on the infected organ itself but on the young, tender leaves which grow out on the stems, and at the ends of the side shoots. The virus, or poison, seems to be carried in the sap to the last-formed tissues (the meristem) and there first of all to make its action evident. The transmission of the disease from plant to plant is easily effected through the hands or tools of the workers when the topping of the plants—to prevent flowers from forming—is being carried out. For this reason, the side shoots of topped plants suffer, as a rule, from mosaic. Diseased leaves are much less valuable than healthy ones, they are unsuitable for the outside of cigars, and they are said to impart an unpleasant, penetrating smell to pipe tobacco.

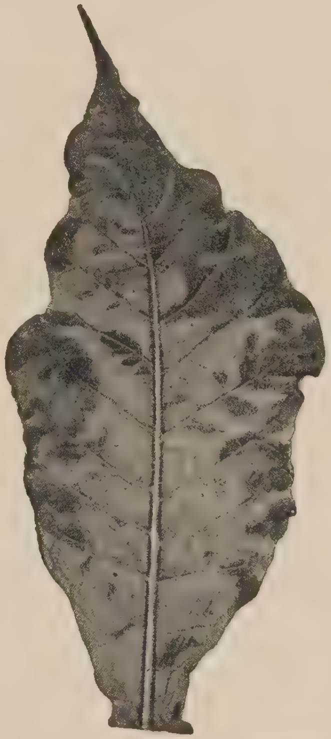

Fig. 394.—Tobacco leaf suffering from Mosaic. (After D. Iwanowski.)

The disease attacks all cultivated varieties of *Nicotiana tabacum*, but is believed not to appear on *N. rustica*.

The cause of this disease has not yet been ascertained with certainty. According to one view it is due to bacteria which are too small to be detected by the microscope or to be demonstrable in cultures (Mayer, 1886; Koning, 1898). Other investigators favour a living infective material, "contagium vivum fluidum," closely bound up with the protoplasm of the cells (Beijerinck, 1898), forming a kind of "mycoplasm." A third view refers the cause to peculiar accumulations of protoplasm, reminiscent of parasitic amœbæ, found in the vicinity of the cell nucleus or in immediate contact with it—a sort of " contagium fixum " (Iwanowski, 1903) ; these bodies have recently been named *Strongyloplasma Iwanowskii* (Palm, 1922). Finally, the presence of any parasitic causal organism is denied and the disease is regarded as being the consequence of certain disturbances in the normal metabolism of the plant, due to high temperature, excessive water supply, damp atmosphere, insufficient nourishment, etc. The result of these disturbing factors is either the over-production of oxydases or peroxydases, due to enzyme activity, or the formation of toxic substances in consequence of external stimuli (Heinzel, 1900 ; Woods, 1902).

This disease, which has long been known in tobacco-growing countries, was first investigated in Holland about the middle of the eighties of the last century. Since then much attention has been given to it, not only in Holland but also in France, Russia, North America, etc. On the tobacco fields in the vicinity of Stockholm the disease has reappeared each year for a long time past.

Control Measures :

(1) Seed should only be taken from healthy plants. (2) Seed should be stored in a dry place and not allowed to come in contact with any of the remains of diseased plants. (3) The soil of the plots in which the plants are raised must be free of the disease. (4) The soil should be well drained. (5) The use of fresh dung is inadvisable, but kainit and basic slag are recommended. (6) The mosaic-infected plants should be topped first ; a few days later, after carefully disinfecting the hands and tools, the healthy plants can be topped.

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Another virus disease, known as " Pox," is often present in the same tobacco fields as the above-mentioned disease, sometimes on the same plants. Numerous small, often minute, brown or whitish spots, which dry up and in some cases burst, are found scattered over the whole lamina of the leaf.

Mosaic or virus diseases also occur on a large number of garden plants, particularly in North America. The following have been observed and described: on potatoes (Tomkins, 1926), on sweet potatoes—*Ipomœa batatas*—(Pratt, 1916), on celery (Poole, 1922), on peas (Doolittle and Jones, 1925), on beans (Reddick and Stewart, 1918, 1919), on *Cucurbitaceæ* (Doolittle, 1920, 1924; Gardner and Kendrick, 1923; Walker, 1926), on lettuce (Jagger, 1921; Newhall, 1924), on hops (Salmon, 1923), on blackberries (Rankin and Hockey, 1922), etc.

Peach Yellows, Peach Rosette, and Little Peach

Certain very destructive diseases occurring on peach trees have been known for a long time in America. The effect of these diseases is similar to that caused by *Taphrina deformans* (Berk.) Tul. (p. 270), but the organisms which give rise to them have not yet been discovered.

In the case of **Peach Yellows** the first symptom is the premature ripening—from a few days up to three weeks earlier than usual—of a larger or smaller number of the fruits. In addition, the outside of the fruit bears red spots or blotches and the flesh is marked with reddish streaks. Such prematurely-ripe fruit has usually an insipid, distasteful or bitter flavour. In the same year or in one of the early succeeding ones, a second symptom of disease appears in that the young twigs and leaves develop abnormally. The one-year-old shoots show a tendency

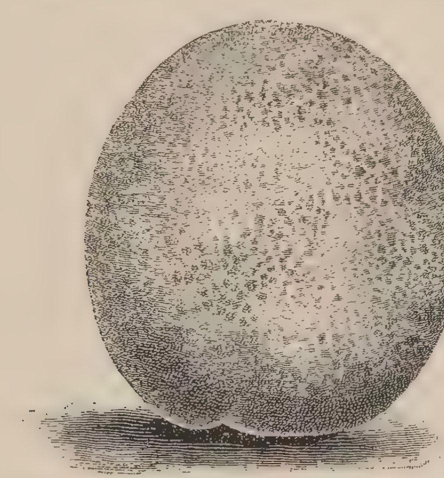

Fig. 395.—Peach Yellows. A premature peach. (After E. F. Smith.)

to branch and form witches' brooms. The leaves become narrow, greenish-yellow, in-rolled, and they continue to grow after the growth has ceased in the rest of the head of the tree. The disease reappears each season with increased intensity as a rule, but it is not equally destructive in all years; in certain seasons up to 25 per cent. of the trees may be affected. From the second or third year of the disease onwards, fruit formation ceases, and in the fourth or fifth year the tree generally dies; trees that are attacked do not recover. A diseased tree in a nursery may be a centre of infection for the neighbourhood as the buds, which carry the disease, may be grafted to healthy stock. It is in this way that the disease is spread and not by pollen or through the soil. By means of buds taken from infected trees, it has been possible to transmit the disease artificially, the period of incubation in such cases varying from eight months to five years.

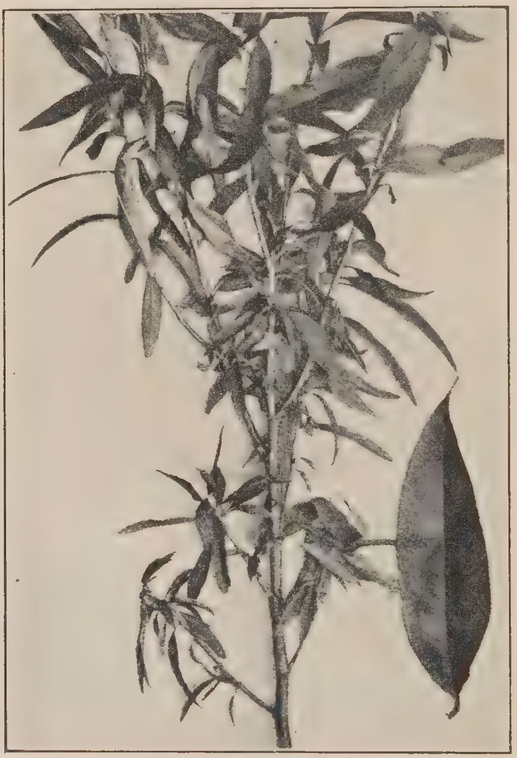

Fig. 396.—Peach Yellows. Typical yellows shoot with one normal healthy leaf at right. (After M. A. Blake, M. T. Cook, and C. H. Connors.)

Mention was made of the disease in Philadelphia as early as 1791; in 1806–07 it appeared in Pennsylvania, New Jersey and Delaware, some years later it reached New York and Maryland, whilst in 1870 it was reported in Canada. Recently the disease has spread widely along the Atlantic Coast from Massachusetts to South Carolina and westwards in Michigan, Illinois, Missouri, and Kansas.

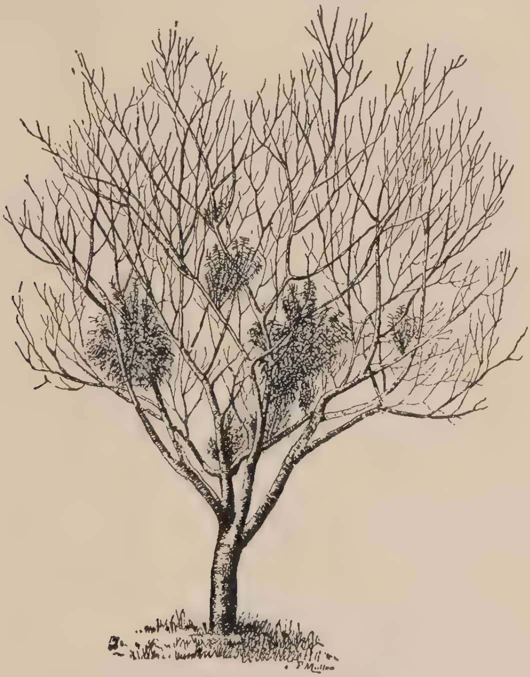

FIG. 397.—Peach Yellows. Diseased tree in the fourth year. (After E. F. Smith.)

" The peach orchards of Pennsylvania contain nearly five million trees. Since 1921 this State has maintained an annual inspection for the eradication of the disease in fourteen of the south-eastern counties comprising over 400 commercial orchards. The records show a definite decrease of infection in this area from 4·5 per cent. in 1921 to 2·21 per cent. in 1923. Such inspections can profitably be made (*a*) just before blooming, (*b*) about July 1, (*c*) a week or ten days before the ripening of the fruit, and (*d*) in late summer." (W. A. McCubbin, 1924.)

Another similar disease, **Peach Rosette,** has been known, particularly in South Carolina, Georgia, and Kansas, for several decades. In early spring the leaf buds grow into compact bunches or rosettes which, although only 2–3 inches long, may contain several hundred very small leaves. The outer, older leaves have inrolled margins and assume a characteristic stiff appearance ; they also fall prematurely. The inner, smaller leaves of the rosettes are yellowish-green or olivaceous in colour.

As a rule, the fruit buds do not develop. The course of the disease is very rapid; an infected tree dies during the winter or in the following autumn. This disease is often present on the same tree as Peach Yellows.

There is, finally, a third disease of peach trees, to which the name **Little Peach** has been given, which has been known in New York and New Jersey since 1893 and in Michigan since 1898. The leaves, which remain small and only grow to about half the normal size, are yellowish-green, and of a drooping appearance: they fall prematurely. The fruit also remains small, only $\frac{1}{2}$–$\frac{1}{3}$ the usual size, somewhat flattened and rectangular in shape, and does not ripen until 10–14 days later than normal. The flavour of the fruit is insipid or even bitter, the flesh is stringy and tough.

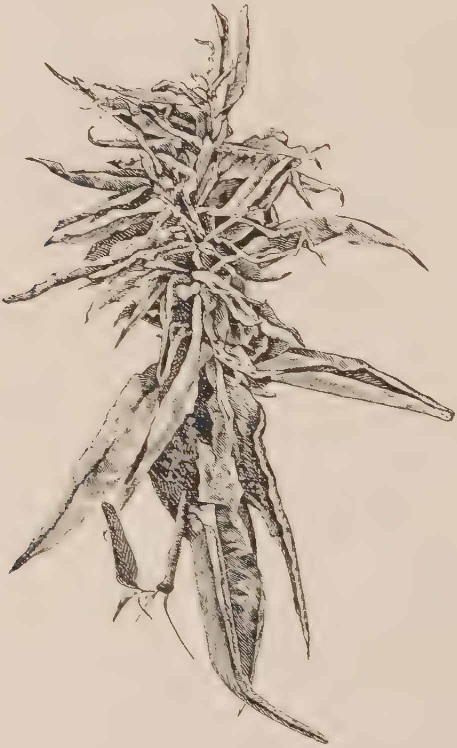

Fig. 398.—Peach Rosette. A single rosette. (After E. F. Smith.)

The above diseases are also said to occur on nectarines, almonds, and apricots; the same, or very similar, diseases attack plums.

Although these peach diseases have been known for a long time, the primary causes have not yet been made clear. Neither fungi nor bacteria have been detected, and consequently the diseases have been classed with the still unexplained mosaic diseases of tobacco, tomatoes, pepper, etc., the cause of which has been referred to a " virus." Recent comparative investigations (Cook, 1921), carried out on healthy and infected leaves in early morning and late evening, have shown that in the leaves of trees suffering from Peach Yellows or Little Peach, the starch manufactured during the day is only incompletely transferred during the succeeding night to other parts of the plant and there used up. Instead of the normal translocation, the starch remains stored in the midrib of the leaf, with the result that the transport of food to the growing tissues is lessened and leads naturally to a check of the growth of the tree. Although this explanation shows the effect of the hidden cause of the disease, it does not disclose the nature of the cause itself.

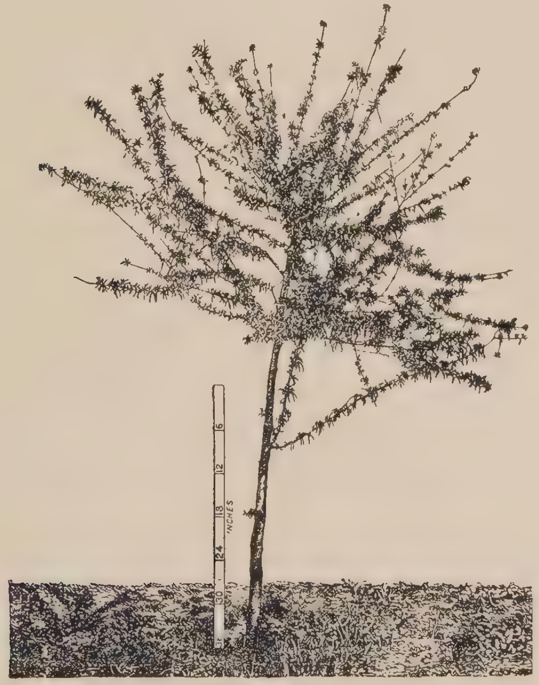

FIG. 399.—Peach Rosette. A diseased tree. (After E. F. Smith.)

To prevent the introduction of these diseases the importation into British Columbia of all peach fruit pits, or seeds for propagating purposes, was prohibited by the Canadian Department of Agriculture (Regulation No. 14), on and after June 22, 1923, from the States of Wisconsin, Illinois, Missouri, Arkansas, Mississippi, and the area east thereof.

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CHAPTER XXII

GENERAL METHODS EMPLOYED AGAINST PLANT DISEASES

THE measures taken to combat plant diseases due to parasitic fungi fall into two groups: (*a*) prophylactic or preventive, and (*b*) therapeutic or curative.

When disease breaks out in agricultural crops such as cereals, roots, clovers, etc., it is clearly much more difficult of control than in the case of garden crops where individual plants, bushes, or trees can often be treated separately. On this account, the measures applicable to cultivated agricultural plants are mainly of a prophylactic nature.

Prophylactic Measures:

1. *The use of Healthy Seed harvested from Healthy Plants.*—In certain cases, as when ergot or smut is present in cereal or grass seeds or when sclerotia occur in the seed of clover, mangel, turnip, mustard, etc., an ocular examination (made by the farmer himself, or at a seed-testing station) shows that the seed is not disease-free.

In many cases, however, a microscopical examination fails to reveal any trace of spores or mycelium although experience may have shown clearly that the disease is seed-borne. This occurs, for instance, with cereal seed and such diseases as smut or rust. To be protected against diseases of this nature the seed must be taken from a healthy crop. Even this precaution is often inadequate because the disease may be latent in the stock for several years until a season arrives in which weather and other external conditions lead to a more or less severe outbreak. Indisputable proof of this is furnished in the case of some wheats which are resistant to yellow rust and where the seed—whether fully developed or shrunken—produces apparently healthy crops of well-developed grain for several years in succession. If, however, there comes a year in which the weather conditions are especially favourable to the development of the yellow rust fungus, the disease breaks out again to a serious extent. In cases of this kind it is best to make a personal examination of the crop from which the seed is to be obtained, but should this be impossible, then reliable information concerning the health of previous crops should be sought. Seed for roots and leguminous crops should be collected, if possible, from home-grown crops.

2. *Selection of Resistant Varieties and Sorts.*—The different races of the same variety of plant often vary in their susceptibility to disease. This is shown, for example, by potatoes in respect of blight and by wheat in regard to yellow rust, etc. Attention should therefore be paid to local experience gained under similar conditions, as the variety or sort may differ in behaviour from place to place.

On the other hand, there are cases in which little or no variation in susceptibility can be detected in the different strains; this is seen generally in bacterial and *Fusarium* diseases.

3. *Careful Cultivation and Drainage of the Field.*—Experience has shown that excessive moisture as well as drought renders the plants more susceptible to disease, so that efforts should be made to avoid either of these extremes.

4. *The Avoidance of Fresh, Unrotted Dung.*—Infection may be carried to young plants in fresh dung.

5. *The Destruction of Diseased Shoots, Leaves, and Roots.*—Disease is often carried in infected plant débris; the destruction of the latter by digging deeply into the soil or by burning should be practised.

6. *Frequent Inspection of Stored Root Crops.*—Any diseased roots that are removed should not be thrown on the manure heap. The infected dung may lead to an outbreak of disease in another crop.

7. *Starving out the Parasite.*—If a parasitic disease occurs in a field the fungus can often be starved out by discontinuing, for several years, to grow any of the plants upon which it lives. This method is especially applicable to root parasites such as " club root " in plants of the cabbage family, *Rhizoctonia* of carrots, etc.

8. *Removal of Plants that carry the Infection to Cultivated Crops.*—Such a plan is advisable when dealing with the heterœcious cereal rusts. Host plants which bear the cluster-cup stage—varieties of *Berberis*, *Mahonia*, *Rhamnus*, and *Anchusa*—should not be permitted to grow within 50–100 yards of fields of corn. Similarly, such plants as couch grass (*Agropyrum repens*), cocksfoot (*Dactylis glomerata*), etc., upon which the same varieties of rust occur, should be removed.

9. *Disinfection of the Seed.*—This can be carried out in several ways:

(*a*) Treatment with Copper Sulphate solution. See p. 123.

(*b*) Treatment with Bordeaux Mixture. See p. 123.

(*c*) Treatment with Formalin solution. See p. 124.

(*d*) Treatment with Corrosive Sublimate (mercuric chloride) solution of a strength of 1 in 1,000 (4 ozs. to 25 galls. water). The solution is made by stirring the weighed quantity of corrosive sublimate with the proper amount of cold water in a wooden (not metal) vessel. Such a solution has been successfully used for the disinfection of scabby seed potatoes. The potatoes were placed in the solution for 1½ hours and afterwards washed in running water for 15–30 minutes.

(*e*) Hot-water treatment. Particulars of the method are given on p. 128. Various appliances have been designed to facilitate the operation; one form, due to Appel and Gassner, allows of the treatment being completed within 12–15 minutes.

(*f*) Treatment with Uspulun or Germisan. See p. 124.

(*g*) Dry disinfection. See p. 125.

Therapeutic Measures:

When disease breaks out in growing crops, such as cereals, roots, or fodder plants, it is clearly not possible to attempt to treat individual plants as is done with trees or shrubs in a garden. The crop must be dealt with as a whole by the application of some material that will kill the fungus. The following are the chief materials used as fungicides:

1. *Powder or Dust Fungicides.*—(*a*) Finely-divided sulphur. The action of sulphur as a fungicide, although not yet clearly understood, is to check the development of disease in cases where, as with the powdery mildews, the fungus has an external, or surface, mycelium. The sulphur should be pure and in the form of a fine powder; it is then more active and adheres to the leaves. Sulphur was first used in Southern Europe in the middle of last century against the vine powdery mildew, and very large quantities of it are still employed in vineyards for this purpose. Sulphur is also used against other powdery mildews, such as those of peas, hops, etc., as well as in the greenhouse against rose mildew.

(*b*) Copper dusts. Most of these dusts consist of finely-powdered copper sulphate (sometimes wholly or partially dehydrated) together with some other material such as oxide or hydrate of lime, or inert substances, such as talc, which are claimed to make the dust adhere better to the leaves. Combined copper-sulphur dusts have also been used. The composition of the copper-containing dusts is very variable, so that it is desirable to make some preliminary trials to ascertain their value for the object in view before using them on a large scale.

Various forms of machines for the application of dust fungicides are now on the market and information concerning them should be obtained, if possible, from those who have had experience of their performance.

2. *Liquid Fungicides.*—In the eighties of the last century, liquid fungicides were largely used in France against diseases of the vine. Shortly afterwards they were tested in England and North America.

The extensive trials carried out in the latter country have led to the general employment of fungicidal liquids in the treatment of many different diseases.

One of the most important of the liquid fungicides is Bordeaux mixture. To prepare a 1 per cent. Bordeaux mixture, 1 kg. (or 10 lbs.) of pure commercial copper sulphate in fine crystals (which dissolve more quickly) is dissolved in 50 litres (or 50 galls.) of water in a wooden tub. The copper sulphate dissolves easily if placed in a piece of sacking and suspended in the water, or a little warm water can be used to assist solution. The other constituent of Bordeaux mixture—milk of lime—is made from 1 kg. (or 10 lbs.) of freshly-burnt lime which is slaked and then made up to 50 litres (or 50 galls.). The milk of lime is strained through a piece of sacking into a tub capable of holding 100 litres (or 100 galls.) and the 50 litres (or 50 galls.) of copper sulphate solution is poured slowly into the milk of lime, the mixture being stirred. A blue, gelatinous precipitate is formed and remains in suspension for a considerable length of time if the Bordeaux mixture has been prepared in the proper manner.

When the Bordeaux mixture has been made it may be tested with red litmus paper. If the paper turns blue the fungicide contains lime in excess, but the reddening of the paper indicates excess of copper sulphate and the mixture is unsafe to use.

Bordeaux mixture must be applied when fresh, within 12 hours preferably, and it should be kept agitated during spraying so that the precipitate does not settle. It has been recommended to add sugar to Bordeaux mixture so that the fungicide may be kept for a longer time ; it is better, however, to make only the quantities needed for the day's spraying.

To increase the spreading and adhesive properties of Bordeaux mixture, various substances have been used ; the two which give the best results, generally, are gelatine (glue) and casein. The latter has been recommended in France (Vermorel and Dantony, 1919) and the following recipe given : 100 gr. commercial casein, in a fine powder, is mixed with 1 litre of milk of lime (about 100 gr. lime to 1 litre water) and well stirred. Within a short time the casein dissolves and the solution can be used at the rate of 1 litre to 100 litres of freshly-prepared Bordeaux mixture (Müller-Thurgau, 1919).

For the application of Bordeaux mixture and other spray fluids a large number of appliances, from the small knapsack sprayer to the powerful spraying machine, are available. It is very important in the case of Bordeaux mixture, and most of the other fungicides, that the spray should be applied in a fine, misty form so that the leaves may be thoroughly covered but not excessively wetted. With proper spraying appliances the addition of gelatine or casein is usually not necessary.

When the Bordeaux mixture comes in contact with the mycelium of the fungus the latter is killed; spores of fungi are also prevented from germinating on leaves that have been properly sprayed with Bordeaux mixture.

Other liquid fungicides which find application are copper carbonate, ammonium copper carbonate, lime sulphur, liver of sulphur, etc. Solutions of these materials are used for purposes similar to those for which Bordeaux mixture is employed.

INTERNATIONAL ACTION FOR THE CONTROL OF PLANT DISEASES *

Outbreaks of fungous diseases on cultivated plants cause heavy losses each year in every country in the world. In some cases these losses amount to enormous sums, as the following figures indicate. In 1891, Germany is said to have lost about 100,000,000 dollars owing to cereal rusts, whilst wheat rust in the United States is estimated to have caused a loss of about 67,000,000 dollars. The yearly loss caused to the world-crop by rust is placed at 250,000,000 dollars. In the United States the annual loss through late blight of the potato (*Phytophthora infestans*) is estimated at about 30,000,000 dollars.

The powdery mildew of the vine (*Oidium Tuckeri*) was first noticed in Europe (England) in 1845, and by 1851 it had reached every European vineyard. The downy mildew of the vine (*Plasmopara viticola*) made its first appearance in Europe in 1890, and within ten years it was found in all the European countries where grapes are grown. In 1895, this last disease caused a loss of about 12,000,000 hectolitres of wine in Hungary. The world's annual loss from diseases of the vine is estimated at about 2,500,000,000 dollars.

The chestnut blight (*Endothia radicalis*), first noticed in 1904 in North America (New York), and since common in many chestnut-growing states of that continent, caused a loss of more than 25,000,000 dollars in 1925.

The American gooseberry mildew (*Sphærotheca mors-uvæ*), known in North America since 1834, appeared in Europe (Ireland and Denmark) about 1900, having been found in South-East Russia in 1890. This mildew spread in a few years over all the countries of Europe and led to a reduction, or, in some cases, the abandonment of gooseberry cultivation.

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* This section is based upon the paper, "An Effective Control of Plant Diseases—A Great Economic World Problem." Jakob Eriksson, *Phytopathologische Zeitschrift*, Bd. I., Heft 3, 1929.

The white pine blister rust (*Peridermium Strobi*) made its entry into Europe about 1880 on seeds and young seedlings of *Pinus Strobus* imported from Russia (from Switzerland on *Pinus Cembra*). During the first decennium of the present century the disease spread over North America, being carried from German and French nurseries on imported seedlings ($2\frac{1}{2}$–$4\frac{1}{2}$ millions every year). On account of this disease one of the most valuable trees in the eastern states of North America is seriously threatened with extinction.

In 1927 it is estimated that the annual losses in Switzerland amounted to 10,000,000 francs from rusts, 1,500,000 francs from smuts, and 15,000,000–20,000,000 francs from potato diseases. A low estimate places the total loss to Swiss agriculture from fungous diseases at 70,000,000–90,000,000 francs per annum (Gäumann, 1927).

The idea of international collaboration in the control of the most destructive diseases of cultivated plants was first made public in 1890 in Vienna (Sorauer, Eriksson) and was brought forward at many subsequent international meetings, from Paris in 1900 to Wageningen in 1923 (Eriksson). The International Pathological Congress held in Paris in 1912 favoured the idea, and at the request of the General Assembly of the International Institute of Agriculture an International Phytopathological Conference was arranged in Rome in 1914. At this Conference the principles upon which international collaboration should be based were discussed and efforts were made to organize an International Phytopathological Convention. The outbreak of the world war prevented further steps being taken, and it was not until certain points in the Convention had been modified at the conferences in Rome (April, 1926) and Buenos-Ayres (June, 1926) that it was accepted by almost all countries, European and non-European, belonging to the Rome Institute. Legislative measures were then undertaken to prevent the spread of destructive plant diseases from country to country.

The issue of certificates and the examinations at the frontier do not, however, in many cases, prevent the spread of disease from one land to another owing to the difficulty, perhaps impossibility, of detecting, by microscopical examination, the organism responsible for the disease. This is seen with such diseases as *Pseudomonas Apii* and *Phoma apiicola* on celery; *Bacterium lacrimans*, *Plasmopara Cubensis* and *Colletotrichum lagenarium* on cucumber; *Uromyces appendiculatus* and *Colletotrichum Lindemuthianum* on bean; *Peronospora Schleideni* on onion; *Pseudomonas Syringæ* on lilac; *Peronospora sparsa* on rose; *Cronartium ribicola* and *Puccinia Ribis* on currant, and very many others.

The only means of preventing the entry of diseases of this class is for the seller to certify that the material which is being sent comes from a disease-free stock which has been systematically inspected.

The case for the establishment, in Europe, of an International, or at least an Inter-European, Institute for Phytopathological Research and the drawing up of a programme of work have been urged by the author on many occasions, but so far without result. This position contrasts sharply with the advances which are being made in the improvement of human sanitary conditions. In view of the enormous annual losses in the world's crops it would be thought that combined efforts would be made to cure the destructive diseases which are responsible for these losses.

APPENDIX

TABLE OF THE FUNGOUS DISEASES ARRANGED ACCORDING TO THEIR HOST PLANTS

1. Cereals

Wheat (*Triticum vulgare*): Red Grain 22, Black Chaff 22, *Pyroctonum sphæricum* 67, *Sclerospora graminicola* and *Scl. macrospora* 115, Bunt or Stinking Smut 120, Loose Smut 126, *Urocystis Tritici* 139, Black (Stem) Rust 149, Brown Rust 158, Yellow Rust 160, Typhula Disease 259, *Erysiphe graminis* 275, Snow Mould Disease (Seedling Blight) 307, *Giberella saubinetii* 310, Sooty Mould (Blight) 325, *Mycosphærella exitialis* and *M. basicola* 332, Take-all (Foot Rot) 332, *Gibellina cerealis* 337, *Helminthosporium sativum* 342, Leaf Spot 343, *Leptosphæria exitialis* 344, *Septoria Tritici* 428.

Rye (*Secale cereale*): Red Grain 22, *Tilletia Secalis* 125, Stripe Smut 139, Black Rust 149, Brown Rust 156, Yellow Rust 160, Crown Rust 165, *Erysiphe graminis* 275, Snow Mould Disease (Seedling Blight) 307, *Giberella saubinetii* 310, Ergot 312, Sooty Mould (Blight) 325, *Mycosphærella exitialis* and *M. basicola* 332, Take-all (Foot Rot) 332, *Helminthosporium sativum* 342, Leaf Spot 343, *Septoria Tritici* 428, *S. Secalis* 429, *Marssonia Secalis* 433.

Barley (*Hordeum sativum*): Red Grain 22, Damping-off Disease 69, *Tilletia panicii* and *T. Hordei* 125, Loose Smut 128, Covered Smut 130, Black (Stem) Rust 149, Yellow Rust 160, Dwarf Leaf Rust 169, Typhula Disease 259, *Erysiphe graminis* 275, *Giberella saubinetii* 310, *Mycosphærella exitialis* and *M. basicola* 332, Take-all (Foot Rot) 332, Leaf Stripe 338, Net Blotch 341, *Helminthosporium sativum* 342, Leaf Spot 343, *Septoria Passerini* 429, *Marssonia Secalis* 433.

Oats (*Avena sativa*): Loose Smut 131, Covered Smut 133, Black (Stem) Rust 149, Crown Rust 165, American Crown Rust 167, *Erysiphe graminis* 275, *Læstadia microspora* 332, *Helminthosporium Avenæ* 342, Leaf Spot 343, *Leptosphæria avenaria* 350, Grey Spot 478.

Maize (*Zea Mays*): *Pseudomonas Stewarti* 22, Corn Blight 22, *Peronospora Maydis* 95, Smut 133, *Ustilago Fischeri* and *U. Reiliana* 134, *Giberella saubinetii* 310, *Diplodia Zeæ* 310, *Chromosporium Maydis* 434, Black-Bundle Disease 458, *Fusarium maydiperdum* 467.

Millet (*Panicum miliaceum*, etc.): *Ustilago Panici-miliacei* 136, *U. Rabenhorstiana* and *U. Digitariæ* 137.

Sorghum (*Sorghum vulgare*, etc.): Blight 22, Smuts 135.

Italian Millet (*Setaria italica*, etc.): *Sclerospora graminicola* and *Scl. macrospora* 115, *Ustilago Crameri* 137.

2. Grasses

Barren Brome (*Schedonorus*): Bulrush Fungus 310.

Bent Grass (*Agrostis*): *Tilletia decipiens* 125, Black (Stem) Rust 150, *Puccinia coronata* 167, Bulrush Fungus 310, *Phyllachora graminis* 323.

Brome (*Bromus*): *Ustilago bromivora* 138, *Puccinia bromina* and *P. Symphyti-Bromorum* 160, Crown Rust 165, *Erysiphe graminis* 276, Bulrush Fungus 310, Ergot 313, *Helminthosporium Bromi* 342, *Septoria Bromi* 429.

Cocksfoot Grass (*Dactylis*): *Aplanobacter Rathyi* 23, *Tilletia striæformis* 125, *Uromyces Dactylidis* 197, Bulrush Fungus 310, Ergot 313, *Phyllachora graminis* 323, *Mycosphærella recutita* 332, *Dilophia graminis* 336, *Glœosporium Dactylidis* 433.

Couch Grass (*Agropyrum*): Crown Rust 165, *Erysiphe graminis* 276, Bulrush Fungus 311, *Septoria Agropyri* 429.

Distichlis: *Puccinia subnitens* 180.

Dogstail (*Cynosurus*): Black (Stem) Rust 150.

False Brome (*Brachypodium*): Ergot 314.

False Oat (*Arrhenatherum*): *Bacterium moniliformans* 23, *Ustilago perennans* 137, Black (Stem) Rust 151, *Puccinia Arrhenatheri* 171, *Erysiphe graminis* 275, Ergot 313.

Feather Grass (*Stipa*): *Puccinia Thymi-Stipæ* 192.

Fescue (*Festuca*): *Tilletia striæformis* 125, Black (Stem) Rust 150, *Puccinia Phlei-pratensis* 156, Crown Rust 165, Ergot 313.

Foxtail (*Alopecurus*): Black Stem Rust 149, Crown Rust 165, *Dilophia graminis* 336.

Hair Grass (*Aira*): Black Stem Rust, 150, *Claviceps microcephala* 315.

Holy Grass (*Hierochloa*): Black Stem Rust 150, Ergot 313.

Lyme Grass (*Elymus*): *Ustilago hypodytes* 138, Black (Stem) Rust 149.

Mat Grass (*Nardus*): *Claviceps microcephala* 315.

Meadow Grass (*Poa*): *Tilletia striæformis* 125, Black (Stem) Rust 150, *Puccinia Poarum* and *P. Poæ-alpinæ* 172, *Uromyces Poæ* 197, *Erysiphe graminis* 275, Bulrush Fungus 310, Ergot 313, *Septoria Tritici* 428.

Milium Grass (*Milium*): *Tilletia striæformis* 125, Black (Stem) Rust 149, *Puccinia Milii* 172, Bulrush Fungus 310, Ergot 314.

Molinia Grass (*Molinia*): *Puccinia Brunellæ-Moliniæ* 192, *Claviceps microcephala* 315.

Quaking Grass (*Briza*): Ergot 313.

Reed Poa (*Glyceria*): *Ustilago longissima* and *U. hypodytes* 138, Crown Rust 165, *Claviceps Wilsoni* 315.

Reed (*Phragmites*): *Ustilago grandis* and *U. hypodytes* 138, *Puccinia Phragmitis* and *P. Magnusiana* 172, *P. Isiacæ* 180, *Claviceps microcephala* 315.

Ribbon Grass (*Phalaris*): *Puccinia coronata* 167, Ergot 313.

Rye Grass (*Lolium*): *Tilletia Lolii* 125, *Urocystis Bolivari* 140, Black (Stem) Rust 149, Crown Rust 165, American Crown Rust 167, *Typhula graminum* 259, Ergot 313, *Glœosporium graminum* 433.

Small Reed (*Calamagrostis*): Black (Stem) Rust 150, Crown Rust 165, *Puccinia coronata* 167, American Crown Rust 167, Bulrush Fungus 310, Ergot 313.

Soft Grass (*Holcus*): *Tilletia Holci* and *T. striæformis* 125, *Puccinia holcina* 160, Crown Rust 165, *Puccinia coronata* 167, Bulrush Fungus 310, *Dilophia graminis* 336.

Timothy Grass (*Phleum*): *Tilletia striæformis* 125, *Puccinia Phlei-pratensis* 156, Bulrush Fungus 310, *Dilophia graminis* 336.

Vernal Grass (*Anthoxanthum odoratum*): *Puccinia Anthoxanthi* 172, Bulrush Fungus 310, Ergot 313.

Yellow Oat Grass (*Trisetum*): *Puccinia Triseti* 160.

Sundry Grasses: *Physarum cinereum* 59, *Spumaria alba* 60, Fairy Rings 244, *Fusarium avenaceum* 466.

3. Root Crops, etc.

Beet (*Beta vulgaris*): Bacterial Disease 11, Dégénérescence Gommeuse 11, Wart Bacteriosis 11, Californian Bacteriosis 12, Beet Yellows 13, Mosaic Disease 13, Seedling Bacteriosis 14, Crown Gall 20, *Urophlyctis leproides* 67, *Rheosporangium aphanidermatus* 67, Damping-off Disease 69, *Phoma Betæ* and *Aphanomyces lævis* 70, Downy Mildew 91, Rust 195, *Typhula Betæ* 260, *Microsphæra Betæ* 297, Heart Rot (Blight) 327, Sclerotinia Rot 386, *Ramularia Betæ* 444, Leaf Spot 451, Girdle Scab 456, Root Rot 472.

Cabbage, Cauliflower, Swedes, Turnip, Rape (*Brassica oleracea*, *B. Napus* and *B. Rapa*): Black Rot 14, White Rot 19, *Bacillus oleraceæ* and *B. brassicævorus* 20, Club Root 52, *Olpidium Brassicæ* 67, Damping-off Disease 69, White Rust 116, *Typhula gyrans* 261, *Erysiphe communis* 276, Root Rot 304, *Mycosphærella brassicola* 331, *Leptosphæria Napi* 349, Sclerotinia Rot 386, Botrytis Disease 404, Black-Leg 424, *Phoma Napobrassicæ* 426, Root Rot 472.

Carrot (*Daucus Carota*): *Bacillus carotovorus* 21, *Plasmopara nivea* 95, *Uromyces lineolatus* 180, Root Canker 348, *Sclerotinia sclerotiorum* 385, Root Rot 472.

Parsnip (*Pastinaca sativa*): *Plasmopara nivea* 95, *Uromyces lineolatus* 180, *Erysiphe communis* 276, *Septoria Pastinacæ* 429.

Chicory (*Cichorium Intybus*): *Puccinia Cichorii* 176, *Erysiphe Cichoriacearum* 276, Sclerotinia Rot 386.

Radish (*Raphanus sativus*): Club Root 55, *Peronospora parasitica* 94.

Horse-Radish (*Nasturtium Armoracia*): *Ramularia Armoraciæ* 444.

Salsify (*Tragopogon porrifolius*): *Cystopus Tragopogonis* 115.

Scorzonera (*Scorzonera hispanica*): Sclerotinia Rot 386, *Cystopus Tragopogonis* 118.

Potato (*Solanum tuberosum*): Bacterial Rot 4, Bacterial Wilt 6, Black-Leg 8, *Bacillus solanincola* 10, Blackening of Veins 10, Corky or Powdery Scab 57, Wart Disease 61, Late Blight 71, Pink Rot 79, Rhizoctonia Disease 257, Leaf Spot 453, Early Blight (*Sporidesmium Solani varians*) 454, Common Scab (*Actinomyces æruginosus*) 457, Silver Scurf 458, Dry Rot 469, Leaf Roll 483, Yellowing 486, Buntwerden 486, Internal Disease 486, Sprain 486.

Jerusalem Artichoke (*Helianthus tuberosus*): *Puccinia Helianthi* 176, *Erysiphe Cichoriacearum* 276, Sclerotinia Rot 384.

Celery (*Apium*): Bacterial Blight 23, *Puccinia Apii* 177, Sclerotinia Rot 386, *Phoma apiicola* 427, *Septoria Apii* 429, *Ascochyta Apii* 430.

Onion (*Allium Cepa*, etc.): Soft Rot 29, Downy Mildew 98, Smut 140, *Puccinia Porri* 175, *P. Allii* 176, *Uromyces ambiguus* 200, Willow Rust 233, *Sclerotium cepivorum* 406.

Asparagus (*Asparagus officinalis*): Damping-off Disease 70, Rust 174.

4. Vegetables

Bean (*Phaseolus vulgaris*, etc.): Bacterial Disease 30, Bacterial Pustule 31, *Bacterium viridifaciens* 31, *Phytophthora Phaseoli* 82, Rust 197, Sclerotinia Rot 386, *Phyllosticta phaseolina* 423, *Septoria leguminum* 429, *Ascochyta Phaseolorum* and *A. Boltshauseri* 430, Anthracnose 436.

Artichoke (*Cynara*) : *Ramularia Cynaræ* 444.
Horse-Bean (*Faba vulgaris*) : *Uromyces Fabæ* 196, Fusarium Disease 469.
Pea (*Pisum sativum*, etc.) : Damping-off Disease 70, *Peronospora Viciæ* 94, *Erysiphe communis* 276, Root Rot 304, *Mycosphærella pinodes* 331, *Septoria leguminum* 429, Fusarium Disease 467.
Rhubarb (*Rheum rhaponticum*) : Crown Rot 24.
Spinach (*Spinacia oleracea*) : Downy Mildew 95, *Ramularia Spinaciæ* 444.
Lettuce (*Lactuca sativa*) : Bacterial Disease 25, Downy Mildew 113.
Purslane (*Portulaca sativa*) : *Cystopus Portulacæ* 118.
Cucumber (*Cucumis sativus*, etc.) : Angular Leaf-Spot 28, Cucumber Wilt 29, Downy Mildew 107, *Erysiphe Cichoriacearum* 276, Anthracnose 438, *Glœosporium oligochæton* 440, Bitter Rot 446, Scab 447, Leaf Blotch 450.
Tomato (*Solanum Lycopersicum*) : Blossom-end Rot 25, Tomato Rot 26, Bacterial Spot 26, Bacterial Soft Rot 27, Bacterial Wilt 27, Grand Rapids Disease 27, Stripe Disease 27, *Bacillus carotovorus* 27, Late Blight 77, Damping-off or Foot Rot 81, *Didymella Lycopersici* 366, *Phoma destructiva* 427, *Ph. alternariaceum* and *Ph. Lycopersici* 428, *Glœosporium phomodes* 439, *Cladosporium fulvum* 448, *Macrosporium Tomato* 460.

5. Forage Plants

(Pulses, etc.)

Birdsfoot Trefoil (*Lotus corniculatus*, etc.) : Downy Mildew 93, *Erysiphe communis* 276, *Mitrula sclerotiorum* 422, *Ramularia Schultzeri* 444.
Clover (*Trifolium pratense*, etc.) : *Olpidium Trifolii* and *Urophlyctis bohemica* 67, Downy Mildew 93, *Uromyces Trifolii-repentis*, *U. Trifolii* and *U. striatus* 196, 197, *Typhula Trifolii* 261, *Erysiphe communis* 276, *Plowrightia Trifolii* 323, *Sphærulina Trifolii* 332, Root Felt Disease 347, Clover Rot 381, *Pseudopeziza Trifolii* 419, *Mitrula sclerotiorum* 422, *Phyllosticta Trifolii* 423, Anthracnose or Scorch 432, *Glœosporium Trifolii* 433, *Macrosporium sarcinæforme* 455, Fusarium Disease 469.
Horse Bean (*Vicia Faba*) : Root Felt Disease 348, Clover Rot 382, *Phyllosticta Fabæ* 423.
Kidney Vetch (*Anthyllis Vulneraria*) : *Uromyces Anthyllidis* 197, *Phyllosticta Anthyllidis* 423.
Lentil (*Lens esculenta*) : *Peronospora Viciæ* 94.
Lucerne (*Medicago sativa*, *M. lupulina*, etc.) : Crown Gall 46, 66, Downy Mildew 93, *Uromyces striatus* 197, *Erysiphe communis* 276, *Pleosphærulina Briosiana* 332, *Leptosphæria circinans* 344, Root Felt Disease 348, Clover Rot 382, *Pseudopeziza Medicaginis* 418, *Pyrenopeziza Medicaginis* 421.
Lupine (*Lupinus luteus*, *L. albus*, etc.) : Damping-off Disease 70, *Cryptosporium leptostromiforme* 433, *Pestalozzia Lupini* 443, Fusarium Disease 469.
Meadow Vetchling (*Lathyrus pratensis*) and Earthnut Pea (*L. tuberosus*, etc.) : *Peronospora Viciæ* 94, *Erysiphe communis* 276, Fusarium Disease 469.
Melilot (*Melilotus arvensis*, etc.) : *Erysiphe communis* 276, *Pseudopeziza Medicaginis* 418.
Sainfoin (*Onobrychis sativa*) : *Uromyces Onobrychidis* 197, *Diachora Onobrychidis* 324, Clover Rot 382, *Pseudopeziza Medicaginis* 418, *Ramularia Onobrychidis* 444.
Sedge (*Carex*) : Sedge Rust 178.
Serradella (*Ornithopus sativus*) : Root Felt Disease 348.
Spurry (*Spergula arvensis*) : *Peronospora obovata* 95, *Puccinia Spergulæ* 176.

Trigonel (*Trigonella*): *Pseudopeziza Medicaginis* 418.

Vetch (*Vicia sativa*, etc.): *Peronospora Viciæ* 94, *Erysiphe communis* 276, *Pseudopeziza Medicaginis* 418, *Ramularia sphæroidea* 444, Fusarium Disease 469.

6. Fruits and Berries

Almond (*Amygdalis communis*): *Puccinia Pruni-spinosæ* 192, *Clasterosporium carpophilum* 354, *Glœosporium amygdalinum* 441, *Cercospora circumscissa* 459.

Apple (*Pyrus Malus*): Bacterial Canker 31, Downy Mildew 87, Juniper Cushion Rust 207, Honey Fungus 242, *Pholiota squarrosa* 243, *Ph. aurivella* 244, White Rot 246, *Polyporus sulphureus* 246, Powdery Mildew 283, Apple Canker 315, Apple Scab or Black Spot 361, Brown Rot 389, *Phyllosticta Mali* and *Ph. Briardii* 423, *Sphæropsis Malorum* 431, Bitter Rot 446.

Blackberry (*Rubus fruticosus*, etc.): *Gymnoconia Peckiana* 209, *Phragmidium violaceum* 215, *Plectodiscella veneta* 371.

Cherry (*Prunus Cerasus*): see under Plum.

Chestnut (*Castanea vesca*, etc.): *Stereum purpureum* 256, Oak Mildew 299, *Mycosphærella castanicola* 358, Bark Disease 374, Ink Disease 376, *Melanconis Modonia* 378, *Phoma endogena* 428, *Diplodina Castaneæ* 431, *Cylindrosporium castanicolum* 442, *Rhacodium cellare* 465.

Currant (*Ribes rubrum* and *R. nigrum*): Currant Rust 176, Sedge Rust 178, Currant Felt Rust 220, Willow Rust (*Cæoma*) 233, *Polyporus Ribis* 251, American Gooseberry Mildew 292, *Nectria galligena* 318, *Mycosphærella Ribis* 357, *Botrytis* 405, *Pseudopeziza Ribis* 421.

Fig (*Ficus Carica*, etc.): *Bacillus Fici* 39.

Gooseberry (*Ribes Grossularia*, etc.): Currant Rust 176, Sedge Rust 178, Currant Felt Rust 221, *Polyporus Ribis* 251, American Gooseberry Mildew 289, European Gooseberry Mildew 300, *Nectria galligena* 318, *Mycosphærella Ribis* 357, *Botrytis* 405, Bitter Rot 446, *Alternaria Grossulariæ* 461.

Hazel (*Corylus Avellana*): Bacterial Canker 33, White Rot 246, *Polyporus sulphureus* 246, *Nectria galligena* 318, Sclerotinia Disease 401, *Pestalozzia Coryli* 442.

Mulberry (*Morus alba* and *M. nigra*): Bacterial Disease 39, Necrosis 40, *Polyporus hispidus* 253, *Nectria cinnabarina* 320, *Mycosphærella Mori* 357, *Septoglœum Mori* 443.

Olive (*Olea europæa*): Oliver Tree Canker or Olive Knot 40, *Polyporus fulvus* 251, Brusca Disease 417, *Cryptoascus oligosporus*, *Coniothyrum Oleæ* and *Hormiscium Oleæ* 418, *Glœosporium olivarum* 440, *Cycloconium oleaginum* 445.

Peach (*Persica vulgaris*) and Apricot (*Prunus armeniaca*): *Clostridium Persicæ-tubercularis* 36, *Taphrina deformans* 270, *Sphærotheca Persicæ* 289, *Clasterosporium carpophilum* 354, *Valsa Prunasiri* 374, Brown Rot 396, *Sclerotinia laxa* 403, *Phyllosticta Persicæ* 423, *Cylindrosporium Padi* 441, Peach Yellows 489, Peach Rosette 491, Little Peach 492.

Pear (*Pyrus communis*): Bacterial Canker 31, Bacterial Blight (Fire Blight or Pear Blight) 33, Juniper Rust 202, Juniper Cone Rust 208, White Rot 246, *Polyporus sulphureus* 246, *Taphrina bullata* 267, Powdery Mildew 285, Pear Canker 315, *Fabræa maculata* 355, *Mycosphærella sentina* 357, Pear Scab 364, Brown Rot 389, *Phyllosticta piricola* and *Ph. pirina* 423, *Septoria piricola* 429, Bitter Rot 446.

Plum (*Prunus domestica* and *P. Cerasus*): Bacterial Disease 34, *Pseudomonas*

Pruni (Bacterial Shot-Hole) 36, *Puccinia Pruni-spinosæ* 192, Honey Fungus 242, *Pholiota squarrosa* 243, *Ph. adiposa* 244, *Polyporus sulphureus* 246, *P. igniarius* 250, *Stereum purpureum* 256, *Taphrina Pruni* 264, *T. insititiæ* 267, *T. Cerasi* 269, *T. deformans* (Peach Leaf Curl) 270, Downy Mildew 286, *Polystigma rubrum* 322, *Plowrightia morbosa* 323, Blight or Die-Back 354, *Mycosphærella cerasella* 357, *Fusicladium Cerasi* 366, Cherry Leaf Scorch 369, *Valsa leucostoma* 373, *V. Prunastri* 374, Brown Rot 396, *Phyllosticta prunicoli* and *Ph. cerasicola* 423, *Ph. Beijerinckii* 423, *Cylindrosporium Padi* 441, Bitter Rot 446, *Chætostroma Cerasi* 465.

Raspberry (*Rubus Idæus*): *Phragmidium Rubi-Idæi* 214, American Gooseberry Mildew 294, *Plectodiscella veneta* 371.

Strawberry (*Fragaria moschata*): Bacterial Disease 40, *Phytophthora omnivora* 89, *Sphærotheca Fragariæ* 295, *Fabræa Fragariæ* 356, *Mycosphærella Fragariæ* 357, *Phyllosticta fragariicola* 423, Bitter Rot (*Melanconium fuliginosum*) 441.

Vine (*Vitis vinifera*): Mal Nero 37, Rogna 38, Tuberculosis 38, Roncet 38, Club Root 56, Downy Mildew 102, Esca Disease 253, *Polyporus igniarius* 254, *Aureobasidium Vitis* 256, Powdery Mildew 277, White Root Rot 351, Black Rot 358, Caucasian Black Rot 360, White Rot 367, Vine Rot 402, Roter Brenner 419, *Phoma flaccida* 428, *Septoria ampelina* 429, *Glæosporium amphelophagum* 434, *Pestalozzia uvicola* 442, *Isariopsis griseola* 459.

Walnut (*Juglans regia*): Bacteria Disease 37, *Gnomonia leptostyla* 371, *Cylindrosporium Juglandis* 442.

Whortleberry (*Vaccinium Vitis Idæa*): *Sclerotinia Vaccinii* 395.

(*Vaccinium Myrtillus*): *Thekopsora Vacciniorum* 237, *Sclerotinia baccarum* 395.

(*Vaccinium uliginosum*): *Thekopsora Vacciniorum* 237, *Sclerotinia dioica* 395.

7. Foliage Trees and Bushes

Alder (*Alnus glutinosa*, etc.): *Polyporus radiatus* 252, *Taphrina Tosquinetii* 270, *Valsa oxystoma* 374.

Amelanchier (*Amelanchier Botryapium*): Juniper Cushion Rust (*Ræstelia*) 207, Juniper Cone Rust (*Æcidium*) 208, Powdery Mildew 286.

Ash (*Fraxinus excelsior*): Olive Tree Canker 42, Seedling Rot 84, *Pholiota squarrosa* 243, *Polyporus hispidus* 253, *Nectria galligena* 318, *Mycosphærella Fraxini* 357, *Venturia Fraxini* 366, *Hysterographium Fraxini* 416.

Barberry (*Berberis vulgaris*): *Æcidium Berberidis* 145, 152, *Æ. graveolens* 172.

Beech (*Fagus sylvatica*): Seedling Rot 83, Honey Fungus 242, *Pholiota squarrosa* 243, *Ph. adiposa* 244, *Ph. aurivella* 244, White Rot 246, *Polyporus radiciperda* 248, *P. fomentarius* 250, *P. radiatus* 252, *P. hispidus* 253, *Microsphæra quercina* 299, *Nectria coccinea* 319, *N. cinnabarina* 320, *Phoma fagina* 428, *Glæosporium fagicolum* 440.

Birch (*Betula alba*): *Melampsoridium betulinum* 235, *Polyporus betulinus* 246, *P. connatus* 247, *P. nigricans* 252, *Taphrina betulina*, *T. nana*, *T. alpina*, *T. turgida*, *T. Betulæ*, *T. Janus* and *T. carnea* 269, *Valsa spinosa* 374.

Bird Cherry (*Prunus Padus*): *Thekospora areolata* 237, *Taphrina Pruni* 264, *Polystigma ochraceum* 322, *Gnomonia padicola* 370, *Sclerotina Padi* 394, Brown Rot 400, *Cylindrosporium Padi* 441, *C. Tubeufianum* 442.

Buckthorn (*Rhamnus cathartica, Rh. Frangula*, etc.): *Æcidium Catharticæ* 166, *Æ. Frangulæ* 167, *Nectria galligena* 318.
Caragana: *Uromyces* 200.
Colutea: *Uromyces* 200.
Elder (*Sambucus*): *Polyporus connatus* 247.
Elm (*Ulmus*): *Spumaria alba* 60, *Collybia velutipes* 243, *Pholiota squarrosa* 243, White Rot 246, *Polyporus fomentarius* 250, *P. hispidus* 253, *Taphrina Ulmi* 270, *Phoma oblonga* 428, *Glœosporium inconspicuum* 441, *Pestalozzia maculicola* 443, *Exosporium Ulmi* 463, Die-back Disease 480.
Eucalyptus: *Ustilago Vrieseana* 138.
Genista: *Uromyces Genistæ-tinctoriæ* 200.
Gleditschia: *Polyporus sulphureus* 246.
Hawthorn (*Cratægus*): Juniper Cone Rust 208, *Gymnosporangium Oxycedri* and *G. tauricum* 209, *Taphrina Cratægi* 270, *Podosphæra Oxyacanthæ* 286. *Venturia Cratægi* 366.
Honeysuckle (*Lonicera*): *Ascochyta Periclymeni* 431.
Hornbeam (*Carpinus Betulus*): Honey Fungus 242, *Taphrina Carpini* 270, *Nectria cinnabarina* 320, *Dermatea carpinea* 416, *Phoma sordida* 428.
Horse Chestnut (*Æsculus Hippocastanum*): White Rot 246, *Polyporus sulphureus* 246, *P. connatus* 247, *Nectria coccinea* 319, *N. cinnabarina* 320, *Mycosphærella Hippocastani* 358.
Laburnum (*Cytisus*): *Peronospora Cytisi* 101, *Uromyces* 200, *Cerathophorum setosum* 448.
Lilac (*Syringa vulgaris*): Bacterial Disease 43, Downy Mildew 85, *Stereum purpureum* 256, *Ascochyta Syringæ* 431.
Lime (*Tilia*): Gnomonia Disease or Anthracnose 372, *Pyrenochæta pubescens* 430.
Maple (*Acer*): Seedling Rot 84, White Rot 246, *Polyporus connatus* 247, *Taphrina polyspora* 270, Powdery Mildew 282, *Nectria coccinea* 319, *N. cinnabarina* 320, *Rhytisma acerinum* 416, *Glœosporium acerinum* 440, *Septoglœum Hartigianum* 443.
Medlar (*Mespilus germanica*): *Gymnosporangium Oxycedri* 209, *Sclerotinia Mespili* 395.
Mountain Ash (*Sorbus Aucuparia*): *Rœstelia cornuta* (*Gymnosporangium tremelloides*) 204, Honey Fungus 242, *Venturia Aucupariæ* 366, *Sclerotinia Aucupariæ* 394.
Oak (*Quercus*): *Cronartium quercinum* 228, Honey Fungus 242, *Collybia velutipes* 243, White Rot 246, *Polyporus sulphureus* 246, *P. igniarius* 251, *P. dryadeus* 251, *Stereum hirsutum* 256, *St. quercinum* 256, *Fistulina hepatica* 256, *Taphrina cœrulescens* 270, *Microsphæra quercina* 298, *Nectria galligena* 318, *Rosellinia quercina* 353, *Clithris quercina* 417, *Phoma quercella* 428, *Glœosporium quercinum* 440.
Oleander (*Nerium*): *Bacterium Savastanoi* 42.
Plane (*Platanus*) *Gnomonia veneta* 370.
Poplar (*Populus*): *Micrococcus Populi* 51, Pine-Poplar Rust 232, *Melampsora Laricis, M. Rostrupii, M. Magnusiana, M. Allii-populina* and *M. Larici-populina* 232, 233, Honey Fungus 242, *Collybia velutipes* 243, *Pleurotus ostreatus* 243, *Pholiota squarrosa* 243, *Ph. destruens* 244, *Polyporus squamosus* 246, *P. connatus* 247, *P. fomentarius* 250, *P. igniarius* 251, *Taphrina aurea* 266, *Nectria galligena* 318, *Venturia Tremulæ* 366, *Didymosphæria populina* 367, *Phyllosticta populina* 423, *Ascochyta Tremulæ* 431, *Glœosporium Tremulæ* 441, *Septoglœum saliciperdum* 443.
Prunus triloba: Brown Rot 400.
Quince (*Cydonia*): *Taphrina bullata* 267, Powdery Mildew 286, *Sclerotinia Cydoniæ* 395.

Rhododendron: *Chrysomyxa Rhododendri* 220, *Venturia Rhododendri* 366, *Sclerotinia Rhododendri* 395, *Pestalozzia Guepini* 443.

Robinia Pseudacacia: *Polyporus sulphureus* 246, *Nectria coccinea* 319, *N. cinnabarina* 320.

Sarothamnus scoparius: *Uromyces* 200.

Sloe (*Prunus spinosa*): *Taphrina Rostrupiana* 266.

Sorbus: *Ochrospora Sorbi* 230, Powdery Mildew 286, *Sclerotinia Aucupariæ* 394.

Spindle Tree (*Euonymus europæus*): *Melampsora, Euonymi-Capræarum* 233, *Oidium Euonymi-japonici* 301.

White Beam Tree (*Sorbus Aria*): *Ræstelia* (*Gymnosporangium tremelloides*) 207, *Sclerotinia Ariæ* 395.

Viburnum: *Ascochyta Viburni* 431.

Wild Service Tree (*Sorbus torminalis*): *Ræstelia* (*Gymnosporangium tremelloides*) 207, *Æcidium* (*Gymnosporangium Oxycedri*) 209.

Willow (*Salix*): Crown Gall 46, Willow Rust 233, *Melampsora Amygdalinæ* 234, Honey Fungus 242, *Collybia velutipes* 243, *Pleurotus ostreatus* 243, White Rot 246, *Polyporus sulphureus* 246, *P. igniarius* 251, *Uncinula Salicis* 282, *Nectria galligena* 318, *Venturia chlorospora* 366, *Rhytisma salicinum* 417, *Cryptomyces maximus* 417, *Phyllosticta salicicola* 424, *Glœosporium Salicis* 441, *Septoglœum saliciperdum* 443.

8. Coniferous Trees and Bushes

Chamæcyparis: *Pestalozzia funerea* 442.

Cryptomeria: *Pestalozzia funerea* 442.

Cypress (*Cupressus*): *Pestalozzia funerea* 442.

Douglas Fir (*Pseudotsuga Douglasii*): *Phoma pithya* 427.

Fir (*Abies excelsa*, etc.): *Chrysomyxa Abietis* 216, Canker or Witches' Broom of Silver Fir 235, *Calyptospora Gœppertiana* 238, *Nectria Cucurbitula* 320, *Herpotrichia nigra* 351, *Dasyscypha calyciformis* 381, *Botrytis Douglasii* 408, *Lophodermium nervisequium* 413, *L. Abietis* 414, *Phoma abietina* 427, *Pestalozzia Hartigii* 442.

Juniper, Common (*Juniperus communis*): Juniper Cushion Rust 204, Juniper Cone Rust 207, *Herpotrichia nigra* 351, *Lophodermium juniperinum* 414, *Pestalozzia funerea* 442, *Exosporium juniperinum* 465.

Juniper, Savin (*Juniperus Sabina*): Juniper Rust 202, *Gymnosporangium confusum* and *G. fusisporum* 203.

Larch (*Larix decidua*): Willow Rust 233, *Melampsoridium betulinum* 235, *Polyporus Pini* 252, *Mycosphærella laricina* 358, Larch Canker 379, *Lophodermium Laricis* 414, *Phoma pithya* 427.

Pine, Common (*Pinus silvestris*): Pine Rust 227, *Peridermium Cornui* 228, *P. Pini* a *acicola* 228, Pine-Poplar Rust 230; Honey Fungus 242, *Polyporus radiciperda* 247, *P. Pini* 252, *Nectria Cucurbitula* 320, *Dasyscypha calyciformis* 381, *Lophodermium pinastri* 412, *Cenangium Abietes* 414, *Phoma pithya* 427.

Pine, White (*Pinus Strobus*): Currant Felt Rust 220, *Peridermium Harknesii* 224, Honey Fungus 242, *Polyporus radiciperda* 248, *P. sistotremoides* 252, *Lophodermium pinastri* 413, *L. brachysporium* 414, *Phoma pithya* 427.

Pine, Sundry Species (*Pinus halapensis*, etc.): *Bacterium Pini* 42, *Chrysomyxa Weirii* 217, *Herpotrichia nigra* 351, *Dasyscypha fuscosanguinea* 381, *Lophodermium pinastri* 412, *Brunchorstia destruens* 415.

Spruce (*Picea excelsa*, etc.): *Chr. Ledi* 217, *Chr. Rhododendri* 220, *Thekopsora areolata* 237, *Nectria Cucurbitula* 320, *Botrytis Douglasii* 408, *Lophodermium macrosporum* 413, *Phoma Pini* 428, *Ascochyta piniperda* 430.

9. ORNAMENTAL PLANTS

Acacia lophantha : *Erysiphe Acaciæ* 277.
Aconitum : *Uromyces Aconiti-Lycoctoni* 199.
Alonsoa : *Phytophthora omnivora* 88.
Alsine : *Peronospora Alsinearum* 102.
Althæa (Hollyhock) : *Puccinia Malvacearum* 181, *Glœosporium Althææ* 440.
Ampelopsis : *Plasmopara viticola* 107.
Anemone : *Urocystis Anemones* 142, *Puccinia fusca* 191, *Æcidium punctatum* 193.
Anoda : *Puccinia Malvacearum* 181.
Antirrhinum : *Puccinia Antirrhini* 192.
Arabis : *Peronospora Arabidis* 102.
Arenaria : *Peronospora Alsinearum* 102, *Uromyces caryophyllinus* 199, *Melampsorella Caryophyllearum* 236.
Asperula : *Peronospora calotheca* 102, *Puccinia Asperulæ-odoratæ* 192.
Aspidium : *Glœosporium filicinum* 440.
Aster : *Phytophthora cryptogea* 81, *Erysiphe Cichoriacearum* 276.
Astragalus : *Peronospora trifoliorum* 93.
Atragene : *Puccinia atragenicola* 191.
Atropa : *Phytophthora erythroseptica* 80.
Azalea : *Exobasidium Azaleæ* 262, *Septoria Azaleæ* 429.
Begonia : *Thielavia basicola* 304.
Betonica : *Ustilago Betonicæ* 138.
Brunella : *Puccinia Brunellæ-Moliniæ* 192.
Buxus : *Puccinia Buxi* 190.
Calendula : Botrytis Disease 409.
Calla : *Bacillus Aroideæ* 48.
Callistemon : *Ustilago Vrieseana* 138.
Calocasia : *Bacillus carotovorus* 48.
Camellia : *Pestalozzia Guepini* 443.
Cattleya : *Bacterium Cattleyæ* 51, *Glœosporium cinetum* 440.
Centaurea : *Puccinia Centaureæ* 192.
Cerastium : *Peronospora Alsinearum* 102, *Melampsorella Caryophyllearum* 236.
Cereus : *Phytophthora Cactorum* 88.
Cheiranthus : *Bacterium Matthiolæ* 47, *Plasmodiophora Brassicæ* 55, *Phytophthora cryptogea* 81, *Peronospora parasitica* 94.
Chrysanthemum : *Bacterium tumefaciens* 45, *Puccinia Chrysanthemi* 188, *Oidium Chrysanthemi* 302, *Phoma Chrysanthemi* 427, *Septoria chrysanthemella* 429, *Ascochyta Chrysanthemi* 430.
Citrus : *Pestalozzia Guepini* 443.
Clarkia : *Phytophthora omnivora* 88.
Cleome : *Phytophthora omnivora* 88.
Colchicum : *Urocystis Colchici* 142.
Convallaria : *Botrytis Pæoniæ* 408.
Coronilla : *Peronospora Trifoliorum* 93.
Corypha : *Pestalozzia fuscescens* 443.
Crocus : *Sclerotinia bulborum* 395, *Rhizoctonia crocorum* 477.
Cucubalus : *Uromyces verruculosus* 200.
Cyclamen : Bacterial Disease 47, *Phyllosticta Cyclaminis* 423.
Dendrobium : *Glœosporium cinetum* 440.

Dianthus : *Ustilago violacea* 138, *Uromyces caryophyllinus* 199, *Phyllosticta Dianthi* 423, *Septoria Dianthi* 429, *Heterosporium echinulatum* 449, *Alternaria Dianthi* 463.
Echinops : *Phytophthora Cactorum* 88.
Euonymus : *Cercosporella Euonymi* 459.
Euphorbia : *Uromyces striatus* 197, *U. Genistæ-tinctoriæ* 200, *U. scutellatus*, *U. excavatus* and *U. tuberculatus* 201.
Ficus elastica : *Glœosporium elasticæ* 440.
Forsythia : *Botrytis* 401.
Fritillaria : *Uromyces Lilii* 200.
Gagea : *Uromyces Gageæ* and *U. Ornithogali* 200.
Galanthus : *Urocystis Galanthi* 142, *Puccinia Galanthi* 190, *Melampsora Galanthi-fragilis* 233, *Botrytis Galanthi* 406.
Gentiana : *Puccinia Gentianæ* 190, *Cronartium gentianeum* 228.
Geranium : *Uromyces Geranii* and *U. Kabatanus* 201, *Sphærotheca* 297.
Gilia : *Phytophthora omnivora* 88.
Gladiolus : *Urocystis Gladioli* 142.
Gypsophila : *Uromyces caryophyllinus* 199.
Hedera : Canker 47.
Helleborus : *Peronospora pulveracea* 102, *Entyloma Ranunculi* 143.
Hepatica : *Urocystis Hepaticæ-trilobæ* 142.
Hesperis : *Plasmodiophora Brassicæ* 55, *Peronospora parasitica* 94.
Hyacinthus : *Pseudomonas Hyacinthi* 48, *Sclerotinia bulborum* 395.
Hydrangea : *Septoria Hydrangeæ* 429.
Impatiens : *Cronartium flaccidum* 228.
Iris : *Pseudomonas Iridis* 50, *Puccinia Iridis* 190.
Lavatera : *Puccinia Malvacearum* 181.
Leucojum : *Urocystis Leucoji* 142.
Lilium : *Uromyces Lilii* 200.
Loasa : *Cronartium flaccidum* 228.
Magnolia : *Pestalozzia Guepini* 443.
Mahonia : *Æcidium Berberidis* 145.
Malope : *Puccinia Malvacearum* 181, *Glœosporium Althææ* 440.
Mamillaria : Botrytis Disease 408.
Matthiola : *Bacterium Matthiolæ* 47, *Plasmodiophora Brassicæ* 55, *Peronospora parasitica* 94.
Maxillaria : *Glœosporium Oncidii* 440.
Melandrium : *Ustilago violacea* 138, *Uromyces verruculosus* 200.
Melocactus : *Phytophthora Cactorum* 88.
Mentha : *Puccinia Menthæ* 192.
Muscari : *Urocystis Colchici* 142, *Uromyces Scillarum* 200.
Myrtus : *Ustilago Vrieseana* 138.
Narcissus : *Puccinia Schröteri* 176, *Heterosporium gracile* 449.
Odontoglossum : *Bacterium Pollacii* 51.
Œnothera : *Entyloma Œnotheræ* 143.
Oncidium : *Bacterium Krameriani* 51, *B. farnetianus* 51.
Ononis : *Peronospora Trifoliorum* 93.
Orchis : *Melampsora repentis* 233.
Ornithogalum : *Urocystis Ornithogali* 142, *Æcidium* (*Puccinia simplex*) 169, *Puccinia Liliacearum* 190.
Oxalis : *Æcidium* (*Puccinia Maydis*) 170.
Pæonia : *Botrytis Pæoniæ* 406.
Panax : *Phytophthora Cactorum* 88.

Petunia : Phytophthora cryptogea 81.
Phœnix : Graphiola Phœnicis 143.
Phyteuma : Uromyces Phyteumatum 200.
Potentilla : Phragmidium Potentillæ 215, *Taphrina Potentillæ* 270, *Sphærotheca* 297.
Primula : Peronospora Œrteliana 102, *Uromyces Primulæ, U. Auricolæ* and *U. integrifolia* 200.
Pulsatilla : Puccinia fusca 191.
Rosa : Bacterium tumefaciens 46, *Peronospora sparsa* 99, *Phragmidium subcorticum* 211, *Ph. tuberculatum* and *Ph. fusiforme* 213, 214, *Stereum purpureum* 256, *Sphærotheca pannosa* 287, *Diplocarpon Rosæ* 410, *Phyllosticta Rosæ* 423, *Septoria Rosæ* 429, *Pestalozzia compta* 442.
Salvia : Ustilago Betonicæ 138, *Puccinia Salviæ* 192.
Saponaria : Ustilago violacea 138, *Uromyces caryophyllinus* 199.
Satureya : Puccinia Menthæ 192.
Sceptrum : Cronartium flaccidum 228.
Schizanthus : Phytophthora omnivora 88.
Scilla : Uromyces Scillarum 200, *Sclerotinia bulborum* 395.
Sempervivum : Phytophthora Sempervivi 89, *Endophyllum Sempervivi* 215.
Sida : Puccinia Malvacearum 181.
Silene : Ustilago violaceæ 138, *Uromyces verruculosus* 200.
Solanum : Wart Disease 63, *Phytophthora infestans* 77.
Solidago : Uromyces Solidaginis 201, *Erysiphe Cichoriacearum* 277.
Spirææ : Podosphæra 286.
Stanhopea : Glœosporium cinetum 440.
Stellaria : Peronospora Alsinearum 102, *Ustilago violacea* 138, *Melampsorella Caryophyllearum* 236.
Strutiopteris : Glœosporium Strutiopteridis 440.
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